

THE LARYNGOSCOPE.

VOL. L

JUNE, 1940.

No. 6

A RESUME OF THE LITERATURE ON NECK INFECTIONS FOR 1939.

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During the last two decades the otolaryngologist who reads his journals has had a splendid opportunity to become neck infection conscious, for a host of excellent articles upon this subject have appeared in our current medical literature. Much of the impetus to this recent interest in infections involving the fascial planes of the neck must be credited to Mosher, who, with his two classics upon this subject, probably furnished the inspiration which prompted most of the work that has appeared since their publication.

The majority of the earlier writers about this condition instructed us regarding the anatomy of the potential spaces in the neck, and each discoursed at length regarding the relations of cervical fascia to the formation and limitation of these cavities. All too frequently when an anatomist writes about anatomy he describes it from the standpoint of pure anatomy, which description to the surgical clinician is often confusing, because it lacks the tie-up with surgical indications and remains just pure anatomy. Too many of the dissertations upon the cervical fascia have fallen into this category and have, I am afraid, left the clinician confused rather than enlightened.

Loré¹ in an excellent article, well illustrated with "three-dimensional" X-ray studies of this region, describes the potential cavities of the neck in a manner that makes the surgical approach to them seem easy and logical. He also stresses the fact that while in cadaver dissection the anatomic structures

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 1, 1940.

and relationships are easily seen and identified as they are encountered, as a rule on the operating table, where the tissues of the neck have been distended and matted together by the infection, the picture is vastly different. The surgeon under such conditions must allow for great displacement and often confusing changes in tissue appearances due to the presence of the abscess.

In the second of two splendid publications on neck infections, Beck² analyzes and tabulates the details of 78 cases of this disease. These cases are all well worked up and both the laboratory and clinical findings are discussed at length. The general symptomatology of neck infections is elaborated and then discussed in detail as it applies to each type of abscess that may be encountered in this area. Case histories are shown only in tabulation, but this is so complete as to make this table almost an atlas of the conditions surrounding this type of infection. At the end of this article is a timely discussion of anesthesia methods and their applications to such cases.

Pond³ reports 10 cases of neck infection with complete case histories and tabulation of the details of each.

Alden⁴ reported 68 cases of neck infection, all of dental origin, with a discussion of the bacteriological findings, mechanism of causation and indications for medical and surgical treatment.

Due to its relationships to important contiguous structures, an abscess in the neck may be followed by serious complications, sometimes remote from the primary focus. The literature this year has been especially rich in the details of this phase of the subject.

The position of the great vessels of the neck is such that they are in direct relationship to the posterior end of the submaxillary space and the lower end of the parapharyngeal space, the jugular sheath being for practical purposes, a continuation of this latter space through the neck into the thorax. Little wonder, then, that an abscess in this region may occasionally erode one or more of these vessels and produce a hemorrhage that unless promptly controlled may result in a fatality. White and Hubert,⁵ writing on parapharyngeal hemorrhage, discuss this complication in detail, again describing the anatomic relationships, with particular mention of damage

to the cranial nerves that transverse this region. Several case reports are offered, one of which I quote in detail because it is so typical and so well illustrates the necessity for prompt recognition of the condition and adequate surgical attention if the patient is to be saved.

"J. C., a boy age $5\frac{1}{2}$ years, was admitted to the Willard Parker Hospital on Jan. 1, 1937, with scarlet fever, nasopharyngitis, pansinusitis and adenotonsillitis. The right eardrum was congested and the left dull. The temperature was 101° F., the pulse rate 120 and the respiratory rate 28. There was marked cervical adenitis on the left side. On the tenth day of the disease, spontaneous rupture of the left eardrum occurred. Three days later there was a bloody discharge from the left ear through the perforation. On the sixteenth day brisk bleeding from the external canal accompanied acute mastoiditis. A mastoidectomy was performed. There was excessive bleeding along the posterior wall of the canal, especially near the tip. On the seventeenth day there were profuse nosebleed and excessive bleeding from the mastoid wound. That night profuse nosebleed recurred. The attacks of nasal hemorrhage were reported to have been controlled with epinephrine packs. These hemorrhages were, undoubtedly, from the Eustachian tube. Twelve days after the mastoidectomy there were epistaxis, bleeding from the mouth and much blood in the vomitus.

"On the following day an operation was performed on the neck. Three large, indurated, markedly inflamed glands were removed. No pus was found. The whole region was a continuous inflammatory mass enveloping the large vessels of the neck. The jugular vein had the appearance of being about to rupture at each filling. The common carotid artery was ligated. The external and internal carotid arteries were massed in the remaining inflammatory tissue. There was a clot of blood in the immediate region of the bifurcation of the common carotid artery which proved to be a false aneurysm. The wound was partially closed. A slight hemorrhage from the nose occurred a few hours later. The day after the operation a sac appeared on the left lateral pharyngeal wall, extending to the region of the left arytenoid cartilage. It was punctured with a large needle and analysis of the fluid removed by aspiration gave negative results. Massaging from below

upward brought about the removal of a considerable quantity of thick blood by suction. There was no further bleeding. The patient was very ill from the time of admission and received six blood transfusions, besides infusions of solution of sodium chloride. A bacteriologic examination revealed staphylococcus aureus in the ear and streptococcus hemolyticus in the mastoid region. Two weeks later the silk ligature was extruded through the wound in the neck. On March 23 a plastic operation was performed on the neck. The patient was discharged on April 16. The adenoids and tonsils were removed 10 months later."

Two of Beck's cases were complicated by secondary hemorrhage but he does not describe them in detail.

When in the presence of an abscess in the neck, either before or after it has been surgically drained, the patient has a chill or a succession of chills with fever, a phlebitis of the internal jugular vein or one of its tributaries must be suspected and the vein should be exposed, inspected and ligated if found to be involved.

Colby Hall⁶ reports eight cases of sepsis following pharyngeal infections and says that there are three routes by which infections may reach the general circulation from the pharynx.

1. *Phlebitic*: To the internal jugular vein by a retrograde phlebitis beginning in one of its tributaries in the pharynx.

2. *Phlegmonous*: Here we are mainly concerned with the parapharyngeal space, from which an abscess may reach the circulation by direct involvement of the veins.

3. *Lymphatic*: In this case the vein is involved secondary to a breakdown of one or more of the perijugular lymphatics.

He describes in detail clinical manifestations of this type of sepsis and advises early inspection of the vein in all cases where sepsis secondary to neck infection may be suspected. Eight cases are reported with details. There were three deaths in this group.

One of his cases is quoted as an example because I am sure that many patients have died and their death certificates have been marked "sepsis of unknown origin" when in reality a

transient infection in the tonsil or pharynx was the beginning of the end.

"Case 7. Thrombophlebitis of the Internal Jugular Vein Following Immediately a Mild Pharyngitis: C. O., white, male, age 24 years. Onset of illness, Feb. 21, 1939. Patient noted a slight soreness of the throat for the first time.

"Feb. 24: Severe chills occurred for the first time. These chills continued each day until admission to the hospital.

"Feb. 28: Admitted to the hospital. Examination: Pharynx and tonsils very mildly infected. So mild, in fact, that in the absence of history they would have been passed up as being normal. There was a moderate tenderness and obliteration of the 'subangular space' on the left side. This was the only possible lead as to the diseased vein. Laboratory: W.B.C., 26,400, 90 per cent polys.

"March 1: Temperature rose to 107°, with dyspnea, cyanosis and pain in the left chest.

"Radiographs: Diffuse patchy infiltration throughout the right lung and a smaller amount in the left lung. Venous ligation and resection. The left common facial and the internal jugular veins were markedly thrombophlebitic, the jugular phlebitis extending from above the facial to the clavicle. The pharyngomaxillary space contained no gross pathology. Microscopic examination of the veins: Acute thrombophlebitis. Culture of the veins: Staphylococcus aureus.

"Postoperative Course: Patient was given sulfapyridine, transfusions, staphylococcus phage and other supportive treatment but succumbed to his extensive pulmonary involvement.

"March 7: Patient died."

Liebowitz and Weinstein report six cases of jugular vein thrombosis following post-tonsillitic cervical abscess. They review the literature and offer an excellent bibliography. Each of these cases is described in detail and no one who reads them carefully should fail to recognize this symptom-complex when he meets it.

Infections of the mediastinum which originate in conditions of interest to the otolaryngologist are of two general types:

1. Those which arise in the cervical region and extend to the mediastinum by gravitation.

2. Those which begin primarily in the mediastinum. These latter infections may be due to: *a.* esophagitis; *b.* suppurative tracheobronchial adenitis; *c.* trauma to the esophagus following: 1. instrumentation; 2. foreign bodies.

In a classic monograph upon mediastinal infections, Ochsner and DeBakey⁸ discuss this condition in all of its aspects. Although this article is written from the standpoint of the thoracic surgeon, there is much in it that is of concern to the laryngologist and the esophagoscopist. The mechanism involved in the causation of this complication is considered in great detail and a careful review of the literature is made, with consideration being given to the opinions particularly of some of the earlier writers upon the subject. These authors discuss the pros and cons of early or late surgical drainage of the mediastinum but lean to complete evacuation of the mediastinal abscess as soon as the diagnosis is definitely established. Details of both the cervical and posterior operations for mediastinal drainage are elaborated with excellent drawings. I am of the opinion that this is one of the best contributions to this subject to date.

Orton⁹ discusses buccopharyngeal and periesophageal abscesses and again stresses the necessity for early recognition and operation.

Westley Hunt¹⁰ writes on periesophageal abscesses and describes the method of X-ray diagnosis of this condition devised by Dr. Frederick Law. He reports two cases in detail, with an analysis of 20 more cases that occurred in New York hospitals. He summarizes by saying, "In the presence of known trauma to the esophagus, any rise in temperature leukocyte count or pain, and particularly X-ray evidence of periesophageal involvement, necessitates an immediate (not the next day) external operation and drainage."

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ATROPHIC RHINITIS IN PLASTIC SURGERY.

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Atrophic rhinitis, a disease which has been known since Biblical times, is characterized by a distinctive fetor, the result of a pathologic change leading to crust formation on the mucous membrane. The Latin name, *rhinitis atrophicans fetida and crustosa or ozena*, is all-inclusive and characterizes the disease.

According to Thomson and Negus,¹ the etiology of this affection may be summarized as:

A. Predisposing Causes:

In some cases the disease may be found in members of the same family, due to: 1. contagion; 2. hereditary; or 3. inheritance of predisposing anatomical characteristics.

B. Exciting Causes:

The cause is unknown. Numerous hypotheses have been advanced, the chief of which are classified in the following way:

a. Structural:

1. Congenital narrowness of the nasal fossae (Berliner, Tillot, Sauvage).
2. Undue patency of the nasal chambers, from the type of skull (Hopmann, Siebenmann, Gerber, J. Wright).
3. Arrest of development of inferior turbinals (Zaufal).

b. Secondary, from:

4. Purulent rhinitis of childhood (Bosworth, Lack, C. A. Parker).
5. Ethmoidal osteitis (Tissier).
6. Rarefying osteitis of inferior turbinalis with secondary alterations in mucosa (Cholewa and Cordes).
7. Accessory sinus disease (Michel).

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Oct. 27, 1939.

8. Focal suppuration in the nose, accessory cavities or postnasal space (Grünwald).

9. Atmospheric desiccation and bacterial invasion (Freudenthal).

10. Deficiency disease.

c. Bacterial and Infective:

11. *Coccobacillus* (Loewenberg).

12. *Bacillus mucosus* (Abel).

13. *Bacillus fetidus ozena* (Hajek).

14. *Pseudodiphtheria bacillus* (Bellfanti and Della Vedova).

15. *Coccobacillus fetidus ozena* (Perez).

16. *Pes Gradenigo bacillus*. From clinical observation (Massei, Perez, Lermoyez).

d. Constitutional:

17. Syphilis.

18. Tuberculosis.

19. Atrophic neurosis (W. Williams, Zarniko, Réthi, Chauveau, Ferreri).

20. A toxic infection, particularly slow in progress, acting especially on the vessels and glands of the pituitary membrane, and possibly associated with tuberculosis.

e. Racial Distribution.

A few of the chief causes of this condition will be discussed herewith:

Atrophic rhinitis is frequently met with in negroes residing in South, Central or North America, whereas examination of negroes in Africa, Oceanica or the West Indies fails to disclose its presence.¹ According to Roy,² this would indicate the infectious nature of the disease.

Suppuration of the accessory sinuses cannot be accepted as the cause of the affection, inasmuch as the sinuses are incompletely developed and rarely found to suppurate at puberty, when atrophic rhinitis is usually seen. In the pres-

ence of suppuration of the sinuses it is difficult to ascertain which is the primary process. It has also been noted that frontal and maxillary sinusitis are seldom associated with atrophic rhinitis, whereas the same process in the sphenoidal and posterior ethmoidal sinuses may do so.

The bacteriological viewpoint has as yet not been clarified. The various organisms, as outlined in the table, may be etiologic factors in the development of the situation; however, it is entirely possible that their presence is secondary. In this way they may be the cause of the ozena.

Females are affected from four to five times as frequently as males. Most cases begin at puberty and few originate after the twenty-third year of age.

Atrophic rhinitis is prevalent among races with a short and broad skull (chameprosopic), like the Chinese, the Japanese and the Semites. It is endemic in Spain, where it occurs in one-third of the cases presenting symptoms of nasal disease.

Clinically, it has been noted in 50 per cent of the cases that ozena is usually seen in noses that have wide nasal chambers. The degree of involvement seems to be in direct ratio to the amount of change in the nasal widening. In those cases in which there is a concomitant septal deviation to one side, it has been found that the pathological changes are decreased on the narrow side. The abnormal width of the nose tends to increase oxidation and evaporation of secretions as the pathologic change in the mucous membrane sets in.

The histology of the condition is described by Wyatt Wingrave³ as: 1. transformation of the columnar, ciliated and special olfactory cells into stratified squamous epithelium; 2. disappearance of the hyaloid basement membrane; 3. the presence of special hyaloid bodies and pigment masses; 4. change in the glands; 5. changes in the lymphoid tissue and blood vessels; 6. changes in the bone. These changes produce an alteration in the secretion, which is a thick mucus filled with cellular elements and swarms of micro-organisms. This viscid secretion stagnates and forms crusts which adhere to the underlying membrane. The secretion, having a low water content, evaporates with great readiness and exerts pressure upon its contiguous membrane, the upper layer of which is

formed by columnar epithelium, which is ciliated. The cilia then disappear and the protective, mechanical function of these is lost. Fibrosis and atrophy then set in, affecting the sensory, secretory and vasodilatory nervous elements. This extension may be followed as far as the sphenopalatine ganglion, which according to Vogel,⁴ gives rise to a marked loss of the ganglia and its parenchymatous structure.

The protective influence of the nose to clean, warm and humidify the inhaled air is thus lost. The lower respiratory tract and the whole organism⁵ show the effect of the breakdown of nasal physiology.

The outstanding symptom of atrophic rhinitis is the exceedingly offensive, penetrating odor which permeates everything in the vicinity of the patient. There is loss of the sense of smell, with insensitivity in many cases to the sufferer of the foul odor. There is complaint of a discharge of crusts, with inability to breathe through the nose. There is usually soreness of the pharynx and associated bronchitis or laryngitis. Complications affecting the ears, eyes or stomach are not infrequent. There is often a general deterioration of the health of these individuals. Because of the social implications, these patients are usually pariahs of society, and emotional and mental sickness results not infrequently.

In the differential diagnosis, hereditary syphilis in young persons must be ruled out. In older patients the presence of a foreign body,⁶ rhinolith, empyema, lupus or suppurative adenoids must be considered. The presence of one-sided pathology should lead to investigation of one of the above conditions.

Mortimer, Wright and Collip⁷ believe that this disease is based upon a predisposition to pathologic anatomical abnormality of the facial part of the skull in growth. They found that cranial dysplasia occurred in patients who were shown to suffer from disturbance of the anterior lobe of the hypophysis. They recognized in monkeys the nasogenital relationship. Estrogenic substances produced a specific response in the conchal mucosa. They treated 31 females and seven males, with results justifying the conclusion that estrogenic hormone insufflation results in a therapy for ozena more effective than any other available until the time of their writing.

No striking changes in histologic structure of the mucosa were noticed in 14 of 21 cases of atrophic rhinitis treated with estrogenic substances, as reported by Eagle.⁸ Twenty-one of their 22 patients were clinically improved and desired a continuation of the treatment.

General medical care has been directed toward the improvement of living conditions, improved diet, with the adjustment of sanitary conditions. Good air, avoidance of dust, alcohol and tobacco are equally important. Many patients are anemic



Fig. 1A and B. Patient before operation; subjective symptoms present.

or may suffer from deficiency diseases. All these must be corrected.

A great number of local applications and irrigations have been employed. Their extent and composition is varied, but the principle is the same. They tend to cleanse the mucous membrane.

Electrolysis has been used in the past⁹ and the local application of the constant or interrupted electrical current is now rarely employed. The galvanocautery, accordingly, has lost favor.

The surgical procedures are all based upon the idea of permanently altering the nasal chambers to decrease their width,

and at the same time to cause as little injury to the endonasal structures as possible.

In 1894, Sanger¹⁰ narrowed the nasal cavities, employing metal obturators within the nose. This method was found to be impractical. Gersung¹¹ used paraffin. This inaugurated a search for suitable implants, either heteroplastic or autoplatic. Paraffin was then inserted into the inferior turbinates. Later it was implanted into the septum; however, it had a tendency to spread the tissues. Spongy beef bone was then used and, later, ivory implants, then tibia or rib cartilage, also celluloid, gutta-percha and fat — all utilized for the



Fig. 2A and B. Appearance after operation, with cessation of symptomatology.

purpose of implantation. These implants were deposited beneath the mucosa or septum, inferior turbinates or floor. Atrophic mucosa, ulceration and fistula resulted in many cases.

Other operative procedures were undertaken in an attempt at correction. The vasoconstrictor fibres were removed by the extirpation of the cervical sympathetic ganglia or the pericarotid plexus¹² and the sphenopalatine ganglion. Wachsberger¹³ points out that the results are disappointing because of the rigidity and fibrosis of the nasal arteries and the difficulty in interrupting all the sympathetic fibres to the nose

which tend to anastomose so completely. Injections into the sphenopalatine ganglion interrupt the sensory, together with the sympathetic fibres. The former's normal secretory function is inhibited, thus increasing the dryness.

Lautenschlager,¹⁴ in 1916, reported a procedure by which he narrowed the nasal cavity. The lateral nasal walls were transposed toward the septum. The maxillary sinus was entered from the canine fossa through the labiogingival incision, the nasoantral wall was broken through anteriorly and

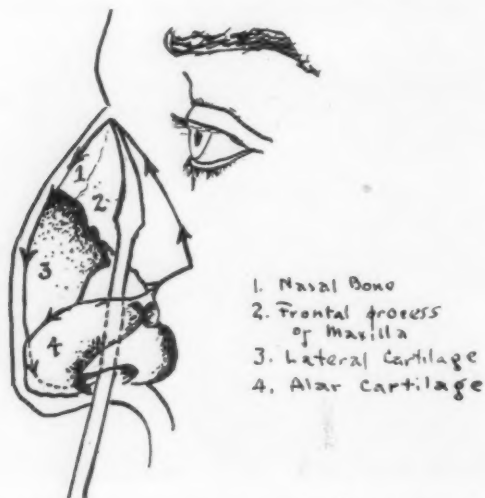


Fig. 3. Undermining of skin on side of the nose along the base of frontal process of the maxilla.

at the base, and forced toward the septum. Adhesions are produced by previous scarification of turbinates and septum. Halle¹⁵ modified this method by endonasal incision.

Wachsberger¹⁶ modified Halle's procedure by performing the entire operation through the nostril. The pyriform crest is displaced, in addition to the nasoantral wall, and thus the degree of the narrowing of the nasal cavity is independent of the size of the antrum. The intranasal approach eliminates the possibility of a fistula between the antrum and the nasal cavity.¹³ Wachsberger further points out that his oper-

ation is successful in persons with small antral cavities as well as in persons with normal antrums. The nasal cavity may be narrowed more effectively if the most anterior part of the lateral wall is mobilized and transposed, which makes the method suitable for advanced cases. The complications encountered were chronic dacryocystitis and empyema of the antrum. Otherwise, results were good.

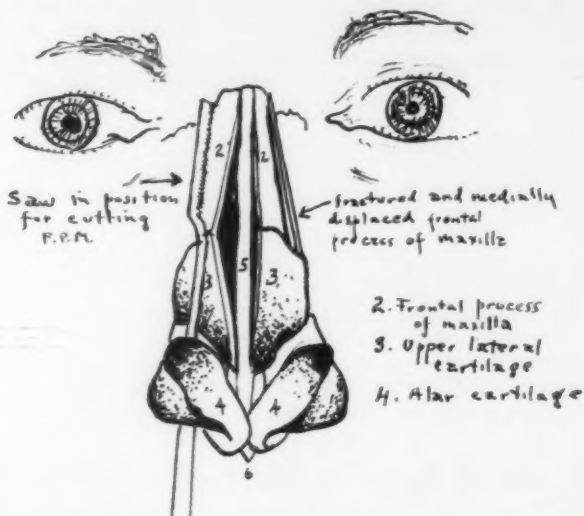


Fig. 4. Line of incision at base of frontal process of maxilla extending up to frontal suture.

The present author suggests a simple method for narrowing the nasal chambers. Five cases of atrophic rhinitis were treated successfully by this means. An incision is made intranasally at the lateral boundary of the pyriform aperture. The skin is undermined bilaterally (see Fig. 3) over the lateral margins of the nose. With a saw, the frontal processes of the maxillary bones are severed from their attachments (see Fig. 4) at the bases. They are then adherent at their sutures at the frontal bones. The fracture is completed manually (see Fig. 5), and the bones compressed medially. By this means the nasal chambers are narrowed considerably. This procedure is one of the steps in rhino-

plasty whereby the nasal bridge is narrowed: A typical case follows:

CASE REPORT.

A young girl, M. C. (see Fig. 1), age 22 years, consulted the author with a history of difficulty in breathing, foul odor and nasal crustations.

Family history negative. Past history: Patient had tonsils and adenoids removed seven years previously, with no improvement in her nasal condition. She had had local treatment to the nose, in the form of ichthyol, glycerine, and iodine preparation tampons. She was treated with tonics; hormone therapy was instituted.

Physical examination revealed a well developed and well nourished, intelligent young lady. There was a marked odor, which was noted at a

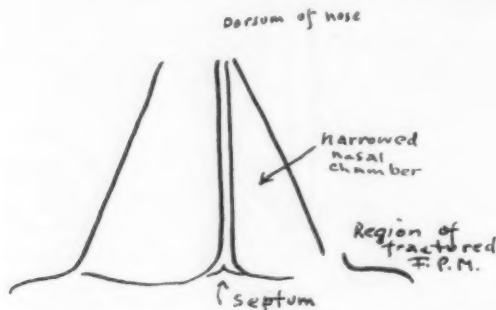


Fig. 5. Left side fractured and displaced medially. Right side before fracture.

distance from her. Her nasal chambers were filled with thick, greenish-brown crusts, which when removed seemed to come out as a cast of the inner nasal chamber. The nasal chambers were then cleansed with sodium perborate solution. The nasopharynx and pharynx were covered with a thick, shining membrane, with bits of this dry mucoid crust attached to it at different points.

Blood and urine examinations revealed normal findings.

After one month of preliminary nasal hygienic treatment, complete rhinoplasty was performed in 1935. The patient's status today (see Fig. 2) is that of marked improvement (intranasally). Physically and from a cosmetic standpoint there is marked improvement.

SUMMARY.

1. Atrophic rhinitis is a disease of unknown etiology. The predisposing and exciting causes have been enumerated.

2. Treatment in the past has been directed toward general improvement of the patient and his surroundings. Local cleansing and irrigations have accompanied the former. Endocrine and vitamin deficiencies have been treated.

3. An abnormal width of the nose is observed in half of the cases in which surgical correction is outlined.

4. A simple narrowing of the nasal chambers by fracturing the nasal bridge bilaterally with the medial displacement, as performed in rhinoplastic procedures, is described. Atrophic rhinitis is no contraindication to rhinoplastic surgery; on the contrary, it is thereby improved.

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THE SYPHILITIC NOSE.

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The pathology in these cases for correction is typical. The nose is pinched and retracted. The dorsum of the nose is completely flat and the tip of the nose is retracted upwards. The skin is intact but has an anemic, glossy appearance. The nasolabial folds are thickened and folded, and the alae are rigidly attached to the aperture pyriformis. The nares, instead of looking downwards in a horizontal plane, look upwards parallel with the plane of the face. The abnormality is due to two factors. The contracting forces exerted by the rigid attachment of the alae to the maxilla by scar tissue and by a marked retraction of the tip of the nose.

The ciliated epithelium of the mucous membrane of the nose is completely destroyed and is replaced by the flat epithelium of cicatricial tissue. The secreting glands are atrophied; the nose is odoriferous and filled with thick, purulent secretions and crusts. There is very little breathing and some anosmia is present. The osseous cartilaginous septum is usually completely destroyed. The entire nose is rigidly encased by fibrotic adhesions to the underlying osseous structures.

It is absolutely futile to attempt to correct these cases by simple rib implantation. The skin is too rigid and will not yield, and the alae are too fixed to permit any adjustment. In order to correct these cases, it is absolutely essential to separate the entire nose from its fibrotic imprisonment to the underlying osseous structures. The nose should then have sufficient lining and covering for the protection and support of the rib transplant.

At present there are two distinct schools for the correction of this pathology:

1. *Buccal Inlay*: Gillies separates the nose from all its underlying scar tissue, and raises it to its normal profile level by means of a detachable dental splint supporting a Thiersch graft on a stent of dental mold, after the principles of Esser's epithelial buccal inlay.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, March 18, 1940.

2. *The Flap Method:* The old Italian and Indian methods in correcting these deformities were not successful because they utilized only one flap. These corrections, remaining without any lining, naturally contracted and the results appeared grotesque. It was not until 1833, when Diffenbach came forward with the idea of giving a lining as well as a covering to these cases that the nose began to take some semblance. Von Mangold, in 1900, was the first to use rib cartilage for the correction of a saddle nose.



Fig. 1.

This thought later stimulated Nélaton, in 1902, to use the principle of cartilage implantation between the flaps to give the nose not only lining and covering but also support which was necessary. With this foundation, many finer and individual modifications have come from Blair, Gillies, Ferris Smith, Esser, Diffenbach, Davis, Ivy, Kazanjian, Sheehan, Figi, New, Straatsma, etc.

While these two schools have greatly advanced the management of this condition, still they present many disadvantages. The Gillies buccal inlay is a highly technical approach and calls for a capable prosthodontist who is highly skilled with

these conditions and must work in harmony with the surgeon. Another disadvantage to the implantation of cartilage is that there is always some danger of going through the thin epidermic lining and thereby exposing these parts to infection. The prolonged hospitalization, with the many stages and after-care, plays a heavy rôle in the economic condition of the patient. This interesting method has never become popular in this country.

The method of choice in this country is the flap method. Any combination of flaps may be used, for fundamentally they



Fig. 2.

are employed for the same purpose. The only objection to this rather interesting approach is that many stages are required, and its subsequent prolonged hospitalization causes great inconvenience to patient and surgeon and places a great economic burden upon the patient.

The following one-stage technique is presented primarily to overcome these shortcomings. While the end-result may not be the best cosmetically, yet its great improvement, which removes, above all, the luetic stigmata, is highly satisfactory to the patient.

Flagg's technique of intratracheal anesthesia has been used exclusively in our series. The apparatus is far away from the zone of operation and the anesthetist is not in the way. A very large costal graft is removed from the seventh, eighth or ninth rib in the usual manner and then shaped to conform to the pattern of the plaster model.

A gingivolabial incision (see Fig. 1, step 1) is made from molar to molar, and the nose is completely separated with periosteal elevators from its marked fibrotic attachments to



Fig. 3.

the underlying osseous structures exactly as in the Gillies approach. The nose is completely released from the maxilla, nasal bones up to the frontonasal suture. It then becomes completely free and movable. It can be brought down somewhat and retroverted if necessary for inspection.

A complete change in gowns and instruments is absolutely essential before proceeding to the nose. Regardless of how carefully and scrupulously the chest and mouth have been prepared aseptically, still it is not a good surgical procedure to proceed to the nose without a complete change. Infection is really the one complication we dread most.

A very deep incision is made in the nasolabial curve, forming a triangle at the lower end into the cheek (see Fig. 1, step 2). This is done on both sides. This step has a twofold purpose: First, it further separates the alae from its imprisonment in these folds; and second, the upturned nares can now be brought down to a normal horizontal plane, leaving a gap between the alae and the cheeks. For the time being, this gap is not sutured.

The columella (see Fig. 1, step 3) is then separated from its attachment to the upper lid and anterior border of septum,



Fig. 4.

and is retracted upwards with a nasal hook and placed out of the way.

With small, heavy, straight scissors, a deep subcutaneous channel is created along the entire dorsum of the nose (see Fig. 1, step 4). This subterranean passage lies between the normal intact skin above and the scarred subcutaneous tissue below.

The rib graft with its columella strut has already been trimmed to conform to this new pattern (see Fig. 1, step 5). Unless there is sufficient room in the tunnel the graft will

slough. This can be attained if necessary by enlarging the tunnel more laterally. There is always enough scarred subcutaneous tissue left in the nose even after complete destruction of the osseous cartilaginous framework to give enough lining for support of this graft.

When the dorsal columella graft is in position, the columella is then brought down and sutured in its normal position (see Fig. 1, step 6).



Fig. 5.

The gap between the ala and the cheek is closed by bringing the triangle flap of cheek (see Fig. 1, step 2) upwards and sutured in manner illustrated in Fig. 1, step 7. Finally, the intraoral incision is closed (see Fig. 1, step 8).

The final result should achieve a raised profile line, nares brought down to a normal horizontal plane, and the nose lengthened.

The transplant is immobilized with adhesive plaster.

Postoperative Therapy: Twenty-five per cent magnesium sulphate, ice compresses to the nose and keeping the nares clean with sterile glycerine are the only postoperative requirements.

For the first 48 hours the patient is given magnesium sulphate for the pain in the chest resulting from removing the rib transplant.

On the third day the sutures on the cheek are removed. On the fifth day, the sutures in the nasolabial fold — columella and the clips in the chest wound are all removed.

This technique is presented because of its simplicity. No highly technical assistants are required, nor the prolonged hospitalization and the multiple stages. This operation is done in one stage and requires only five days' hospitalization. The postoperative care is negligible. The results are satisfactory. The luetic stigmata is completely erased. Figs. 2, 3, 4 and 5 are typical cases illustrating this pathology with correction.

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THE NASAL BIOPSY. A CRITIQUE OF CURRENT METHODS.*

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The methods employed in evaluating histologic studies on the basis of repeated single nasal biopsies are open to serious question in such conditions as hyperesthetic rhinitis, atrophic rhinitis and marked chronic inflammatory processes — since here it is conceivable that faulty deductions often have resulted from the limitations of single biopsy. Very recently, Eagle¹ and his associates used estrogenic substances to treat a series of patients with atrophic rhinitis. In 14 cases, biopsy specimens were obtained from the diseased middle turbinate before and during (or after) treatment. Microscopic study of the specimens failed to reveal obvious changes or definite changes in a single direction. The experience of these investigators substantially corroborated the position previously taken by the writer² in questioning the value of the present method of histologic study of the nasal mucosa in hyperesthetic rhinitis, for single biopsy — rather than multiple — is too restricted to yield sufficient information about the entire nasal mucosa in a variety of pathological states.

From the time the human being is born to the time that he dies, his nose is continuously subjected to the lashings of acute infections, weather conditions, smoke, dust and chemical substances. By recalling that normal nasal mucosa encompasses a pseudostratified columnar epithelium, a basement membrane, and a loose fibroelastic network of connective tissue containing glands and small blood vessels, it is obvious why the human nose undergoes a variety of microscopic changes in adapting itself to ever-changing environmental circumstances. Thus, Hilding's³ morphologic study of the epithelium following variations in ventilation brought up the question whether there are several types of epithelium in the upper part of the respiratory tract, and Latta and Schall⁴ have stated that nasal epithelium may not only be of the simple ciliated columnar type but of the stratified ciliated, the

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Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Nov. 18, 1939.

cuboidal, the stratified cuboidal, the squamous or the transitional type.

The thickness of the basement membrane in the clinically normal human nose varies greatly. For the most part it is definitely well defined, but in some instances it is so delicate as to appear absent; while, conversely, in chronic infections it may be markedly thickened. Is it, therefore, not likely that tissue removed by biopsy from one portion of the nasal mucosa may reveal a thinning or a thickening different from that shown by tissue removed from an adjacent site? And since the epithelium does not always maintain a classical uniformity cannot the same be true here?

Further, under what are regarded as normal conditions the stroma shows a surprising variation in the amount of elastic fibres, ranging from a dense to a sparse formation. Both the middle and inferior turbinates are favorite sites for the taking of biopsy specimens, and their mucosa often varies in thickness so that low cuboidal or squamous variety of epithelium frequently replaces the ciliated epithelium, especially over the anterior tip. Is it, therefore, to be assumed that a nose is clinically abnormal because the microscope displays a deviation from normal? There is, I believe, an urgent necessity for re-examining and re-evaluating the concept of "normal" nasal mucosa.

In chronic inflammatory processes the individual layers of the nasal mucosa show characteristic microscopic changes according to the severity and the duration of the infection. The epithelium and the basement membrane undergo definite thickening, but the thickening is not evenly distributed throughout the entire length of the nasal mucosa. The alterations in the subepithelial layer are governed mainly by the density of the cellular infiltration, and are reflected by changes in the glands and blood vessels.

In allergy the histologic changes are characterized by: 1. edema which involves all the mucosal layers; and 2. an eosinophilic infiltration of the tissues, which together with the edema produces alterations in the glands, the blood vessels and the stroma. Microscopically, hyperesthetic rhinitis displays the characteristics of chronic inflammation. In early allergy the picture is that of simple hypertrophy, but after

secondary infection occurs, hyperplasia develops, with attendant epithelial metaplasia. When chronic infection is superimposed on an allergic process, certain characteristic changes are evolved which represent histologic pictures of both allergic and chronic inflammation of the infectious type. The two changes may vary in individual cases, and even in parts of the same biopsy specimen. Thus, the allergic picture predominates in one case and the infectious in another, or there may be equal or unequal representations of both. Hence, the epithelium may be normal in one area, while in other areas it may appear hyperplastic; the basement membrane absent in one area, slightly thickened in another, and markedly thickened in a third. The edema, eosinophilic infiltration and fibrosis may vary in extent and concentration in the subepithelial layer, while the glands are, accordingly, dilated, cystic, atrophic, compressed or fibrotic.

In histologic studies made before as well as after the use of chemicals, electrocautery, drugs, Roentgen rays, radium and iontophoresis on the nasal mucosa, many investigators have employed the single biopsy method. In view of the fact that the vast majority of biopsy specimens have been obtained from either a chronically diseased nose or an allergic nose, it is self-evident that the mucosa had already undergone pathologic alterations. These alterations I have just reviewed. The problem, consequently, presents itself in a new light: Does a chronically infected nose or an allergic nose constitute a suitable "normal control" for the microscopic study of the effects of chemical, Roentgen rays, radium, cauterizing drugs and ionization? Since pathologic change is characteristic of chronic inflammation and nasal allergy, it is rational to assume that many of the changes seen microscopically after cauterization by any of a number of agents are identical with those that existed before their use.

A specimen removed from a particular site — say, before cauterization — may reveal relatively uninvolved and unaffected nasal mucosa; a specimen from a second site may show more pathologic involvement; while that from a third site may present highly altered nasal mucosa. Thus, two or more specimens taken before cauterization frequently disclose great histologic variations in the tissue. Again, biopsy after cauterization occasionally reveals considerable epithelial insult

and subepithelial change. On the other hand, not infrequently does one observe less pathologic involvement than was seen before cauterization. Has repair of the nasal mucosa begun? Or are variable findings in one and the same turbinate to be allowed for? Further, if biopsies from the same site were removed before and after cauterization, one would encounter destructive changes in the nasal mucosa produced by a punch or scissors which would render any accurate interpretation impossible.

Finally, the microscopic changes encountered in atrophic rhinitis can be divided conveniently into: 1. early changes; and 2. late changes. The early changes are characterized by a chronic inflammatory process; the late changes by a thickening and fibrosis of practically all the arteries. The chronic inflammatory process is often identified by a definite tendency to a squamous-cell type of epithelial metaplasia which progresses along the surface as the pathological change develops. In the later or terminal stages, a dense fibrous tissue, not unlike scar tissue, may be seen directly beneath the altered epithelium. The arteries tend to an obliterating type of endarteritis, the bone undergoes fibrosis and degenerative changes, and the glands are degenerated and replaced by an undifferentiated fibrous tissue.

Paradoxically, a diagnosis of atrophic rhinitis most often is made correctly by the clinician, despite the fact that a biopsy reveals but a chronic inflammatory process. This is one of a number of entities in which the clinical eye is more diagnostic than the microscope. It is only at the time late microscopic changes — chiefly the obliterating endarteritis — occur that the atrophic rhinitis is recognized unequivocally. A chronic inflammatory process, as has been shown, does not lend itself to the single biopsy method, and since the biopsy specimens of Eagle and his associates belonged to the chronic inflammatory process stage of atrophic rhinitis, it is not surprising that their final report left much that was unanswered. It is quite conceivable, however, that multiple biopsies may overcome some of the difficulties I have described. While the single biopsy is too restricted to yield sufficient information about the entire nasal mucosa, perhaps some of the fallacious deductions that have resulted from the limitations of single biopsy may be suppressed by the use of multiple biopsies

before and after the employment of a variety of therapeutic and experimental agents.

CONCLUSIONS.

1. Histologic studies on the basis of single nasal biopsies are open to serious question in such conditions as hyperesthetic rhinitis, atrophic rhinitis and marked chronic inflammatory processes.

2. The information supplied by single biopsy is too restricted to yield sufficient information about the entire nasal mucosa in a variety of pathological states.

3. It is suggested that multiple biopsies before and after the use of a variety of agents on the nasal mucosa may yield sufficient information about the entire nasal mucosa.

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ORAL PLASTIC SURGERY. ABSTRACT.*

DR. HENRY SAGE DUNNING, New York.

I feel that I can cover more ground by showing some lantern slides. Naturally, being an oral man, I am very much interested in cleft lips and palates. We should not say "hare-lip," I think, as the deformity is due to a definite cleft in the lip.

In a complete cleft of the alveolar process the long segment should be pressed over to the short segment and small wire put through, bringing the long segment and the short segment together, giving the child an alveolar arch and closing the anterior bony cleft. Dr. Brophy thought he could do this up to the age of 6 months or a year, but I feel that after the age of 4 months, molding of the bones becomes a little difficult, and the anterior bony cleft should be closed as early as possible.

In repairing a complete cleft of the lips, I feel that, first, the bones should be brought together anteriorly as previously stated. It can be done at 3 or 4 weeks if the child is in good shape, and the lip may be repaired shortly afterwards. In operating on the palate, if the sum of the tissue on both sides of the cleft does not equal the width of the cleft, or more than equal it, you cannot obtain a good result in closing the palate. We may use delayed flaps, and sometimes use Thiersch grafts under the flaps, but we have not found it advisable to attempt to close wide clefts in one operation. If you will consider the muscles of the palate you will realize that if we cannot get a long soft palate we are not going to get very good function. We must have good blood supply and have no infection in order to get a good surgical repair. In large clefts it is better to close them by a two- or three-stage operation, letting a year pass before closing the posterior part of the cleft.

I believe we should loosen the entire tissues of the palate from the gingival margin anteriorly to the tuberosity of the

*Read in full at New York Academy of Medicine, Section on Otolaryngology, Oct. 18, 1939.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Dec. 19, 1939.

superior maxilla posteriorly, loosening it well into the buccal fold. We are careful not to injure the edge of the flap, and after careful denudation we can use mattress and interrupted sutures and get an overlapping of tissue. Cases done in this way do not break down unless some postoperative complication occurs, such as infectious diseases, etc. The Lane operation is not used these days, but some type of the Langenbach is most often employed.

Flaps are loosened on the sides and packed with gauze, which gives much better results than any other method of holding the flaps together. We make full-thickness flaps, sewing one flap over to the other.

In syphilitic clefts we loosen the flaps down to the posterior palatine artery and Thiersch graft the undersurface of the flaps. In these specific cases we get much better repair in this way and generally overlap the edges of the flaps as much as we can.

(Diagram showing loosening up of lateral flaps, denuding the edges to be sutured, and packing with gauze.)

(Demonstration of slides illustrating multiple congenital defects, including double cleft lip and rudimentary tab for an eye; complete cleft into nose and palate; traumatic defects; results of Vincent's stomatitis; ulceration of cheek; osteomyelitis; osteitis fibrositis cystica; chondroma of the cartilage of the nose and maxillary bone; lymphangioma of cheeks; syphilitic defect of palate and nose; rodent ulcer, nose, showing large loss of tissues of the face; macrocheilia associated with pansinusitis; adamantinoma of mandible; bony ankylosis resulting from a fall; prognathism.)

An operation for prognathism after the manner of Blair was illustrated. We section both rami over the entrance of the vessels into the bone at the mandibular foramen and push the jaw back, articulating the teeth as accurately as possible. Careful and firm splinting must follow the operation in order to obtain good results.

In congenital and traumatic lesions of the face there are often associated deformities, and it is very important that

the eye, nose and throat and oral men work together in their repair.

In closing, I should like to say that an oral opening into the sinus should always be closed. An oral opening into the maxillary sinus is generally a dangerous condition as the secretions of the mouth can cause an ascending infection into the nose and accessory sinuses.

33 East 68th Street.

FURTHER ADVANCES IN THE TECHNIQUE OF LARYNGEAL PHOTOGRAPHY.

DR. JOEL J. PRESSMAN and ARTHUR HINMAN,
Los Angeles.

Eighty years have passed since first attempts were made to photograph the larynx. Czermak's¹ pioneer work in this field was reported in 1860, seven years before Lister's paper on antiseptics and only three years after Pasteur propounded the germ theory. The first entirely successful still pictures were made by Thomas French² in 1882, the very year Koch discovered the tubercle bacillus, and by Browne³ in 1883. In 1913, when cinematography was still in its infancy, Chevroton and Vlies⁴ presented, for the first time, motion pictures of the larynx. Until recently this means of physiologic investigation attracted the attention of but very few.

Interest in this subject has been renewed during the past 10 or 15 years, beginning with Clerf's⁵ excellent still pictures, which were taken by a method first introduced by Garel.⁶ These were productive of many important physiological observations. Heatley⁷ developed a splendid technique in 1931, and G. Oscar Russell and Tuttle⁸ in the same year presented most important colored motion pictures taken through a fonopharyngoscope. A year or so later, Francis Le Jeune⁹ succeeded in obtaining the first motion pictures in color with the suspension laryngoscope. The present day enthusiasm which this subject has aroused is largely due to this excellent work of Le Jeune's. Not long after, Gordon New, using the Lynch suspension laryngoscope (as did Le Jeune), presented a noteworthy series of pathological photographs but has not published his method. These latter two contributions did not stress physiologic observations, since the photographs were for the most part taken during general anesthesia, under which circumstances, of course, the opportunities for study of laryngeal physiology are extremely limited.

Our own efforts began about the same time, or possibly a little later than those of Le Jeune. In 1935, Dr. George

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 20, 1940.

Coates presented for us at the meeting of the American Academy of Ophthalmology and Otolaryngology our first motion pictures, taken under local anesthesia through a Jackson laryngoscope.

We subsequently improved our method as we first used it, and a description of the technique appeared in 1937.¹⁴ Since that time the use of the Jackson laryngoscope or modification of it has become increasingly popular. Utilizing this technique, Tremble,¹⁰ in Canada, as well as Holinger and Fishman, who have not as yet published their method, have made fine contributions. Solo, Fineberg and Levine,¹¹ experimenting with 8 mm. film instead of the conventional 16 mm., seem to have suffered an appreciable loss of detail as a result of this deviation but have, nevertheless, contributed their share towards popularizing the use of laryngeal photography.

Gabriel Tucker¹² adapted to the field of cinematography French's technique of obtaining still pictures from a laryngeal mirror. From the standpoint of convenience to the patient this method has no rival. The presence of the epiglottis, however, can be troublesome under the circumstances, since it often hides at least a portion of the larynx.

Lell¹³ has adapted Tucker's method to his own use and cites numerous advantages over the direct laryngoscopic method. Some of these, however, are not tenable and should be refuted so that beginners in this work shall not be unduly prejudiced. For instance, in the matter of obtaining "a relatively more normal picture of the motility of the cords," it has been well established by a comparison of the mirror photographs of French, Garel and Clerf with those taken by direct laryngoscopy, that the appearance of the cords in each instance coincides exactly with corresponding physiological phenomena observed by the alternate direct laryngoscopic method; furthermore, in the matter of expenditure of time it can hardly be held that after the throat is locally anesthetized, the 30 seconds required to pass the direct laryngoscope and obtain photographs through it is excessive. By this latter method there is, contrary to Lell's opinion, no "apparatus to set up" and, therefore, no time at all is required for this preliminary phase. Other advantages of mirror photography which seem important to Lell are more theoretical than real. There is no need to keep the larynx in view while photographing, nor is

it necessary that the larynx be "accurately brought into focus." By the direct laryngoscopic method the larynx automatically stays in view of the camera, which in the final analysis is the important consideration, and focus is no problem at all since this, likewise, is mechanically fixed. For these reasons we fail to see that the arguments Lell puts forward are sufficiently important to wean us away from our direct laryngoscopic method.

We have since 1937¹⁴ been able to improve upon our method reported at that time. Changes in technique since then have resulted in certain advances in addition to those described in the original article. These are:

1. The image is maintained at a fixed position upon the center of the screen.
2. Absence of objectionable glare surrounding the laryngeal structures.
3. Disappearance of the spots of reflected light upon the larynx.
4. Photography in color which very closely reproduces the true color of the larynx.
5. Slow motion (in black and white up to 128 frames per second), which represents a slowing to about one-eighth normal speed at the usual projection rate of 16 frames per second. This, of course, does not compare with the Bell Telephone Co.'s films at 4,000 frames per second, which, however, requires highly specialized technical equipment.
6. A very satisfactory adjustable focus, usually upon the entire depth of the larynx at the same time.
7. Photographs which are well centered.
8. Successful photography in about 90 per cent of the footage of film exposed. This obviously presupposes an adequate exposure of the larynx with the direct laryngoscope.
9. Magnification is adequate and can be increased at will. We have arbitrarily selected our magnification so that it is great enough to show details quite clearly and yet not so large that it precludes the possibility of photographing the entire length of larynges that are larger than average. For

the sake of standardization we use the same magnification in all cases.

It is not to be assumed that the method has no disadvantages as compared with other methods, or that it even approaches entire satisfaction. Its shortcomings are apparent and to us appear as follows:

1. Cocainization is necessary in all cases, whereas in indirect photography it can often be omitted.

2. Direct laryngoscopy is not always a desirable procedure, especially in well trained singers or nervous people, who often hesitate to submit to the procedure lest their voice be harmed, which, of course, should never happen. It is, however, difficult to convince them of this; furthermore, the larynx cannot always be adequately exposed for photographic purposes.

3. In certain diseased larynges, such as severe acute infections, or in exquisitely sensitive pathological larynges, the direct method of exposure may be contraindicated.

4. Extremely brilliant lighting such as is required for the Bell Telephone Co. photographs at 4,000 frames per second¹⁷ cannot at the moment be utilized by the direct method. The inclusion of a stroboscope, however, in the photographic equipment now being used can by the direct method result in color photographs reduced to a speed approaching 24,000 frames a second, or a reduction to 1/1500 normal speed. We have not yet attempted this. The stroboscopic picture, of course, is not a true picture of a single movement. Stroboscopic lighting has already been used and reported upon by Tiffin¹⁸ and Moore.¹⁷

5. There is possibly some objection from the standpoint of physiological study to the stretching of the cords as a result of the anterior "lift" of the laryngoscope; however, comparison of our photographs with those taken by the mirror method would seem to indicate quite definitely that this does not in any appreciable way distort either the appearance or action of the cords, regardless of the type of activity taking place. In one case of weakness of the internal tensor muscles with exaggerated bowing of the vocal cords we felt that the image was possibly distorted, but in no other single instance

did this hold true. Our photographs have otherwise invariably proven to be exact reproductions of the larynx as seen with a mirror. We have made such control observations dozens of times and under all circumstances.

6. The method does not permit photography of the larynx and epiglottis at the same time. The elimination of the image of the epiglottis is, however, often a distinct advantage since only too often it hides the vocal cords, or portions of them. This is especially true when photographing the larynx dur-



Fig. 1. Laryngoscope with light carrier removed, demonstrating its relative length. (A) The light carrier removed from its channel. (B) Hollow loop joining light carriers and containing electric wiring. (C) Channels for light carrier. (D) Channels for fixation prongs on telescopic tube.

ing the production of its sphincter action, as in cough, swallowing and thoracic fixation.

Method: Photography by this method is carried out under local anesthesia as for any direct laryngoscopy. The direct laryngoscope exposes the larynx and through it the photographs are taken. Previously we had used the Jackson-Pilling direct laryngoscope but have now discarded it in favor of the Negus instrument made by the Genito-Urinary Manufacturing Co., of London (see Fig. 1). These instruments are much alike, except that the latter instrument instead of being lighted distally by a single light carrier con-

tains a short double light carrier with larger bulbs, and thus provides a brilliant semiproximal source of illumination (see Fig 1, A).

The light carriers, one on each side of the laryngoscope, are not those provided with the laryngoscope. We have had made a carrier 2.5 cm. long, exclusive of the bulbs but including the socket, and insert into each a $2\frac{1}{2}$ volt Welch-Allyn otoscopic lens-front bulb. These carriers are joined together by a hollow metal loop (see Fig. 1, B), which is standard on the Negus laryngoscope light mount. Within the lumen of this loop electric wiring passes to each light carrier. Standard mounts are wired in series, but we have these changed to

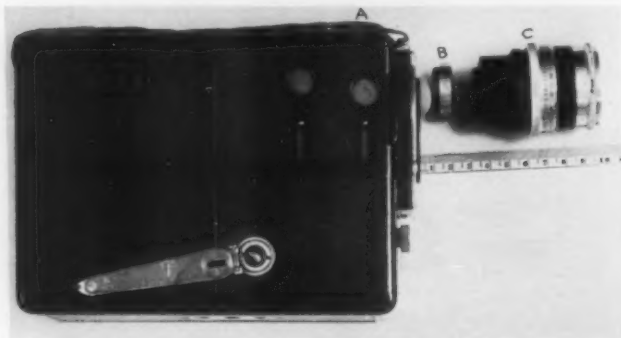


Fig. 2. (A) The camera. (B) Adapter band. (C) Lens.

wiring in parallel. A single source of power thereby brilliantly lights the two bulbs. House current of 110 volts is passed through a rheostat, which in turn is turned as high as possible without burning out the bulbs. Our own rheostat has a "stop" at this point, built into it for us by our instrument maker, the L. C. Isom Co.

The interior of the laryngoscope is especially treated, first by sand blasting and then by a process of black-nickeling with boiling hot "liver of sulphur." This does away with almost all reflection of light from the walls of the laryngoscope so that no glare surrounds the photograph of the larynx.

Any good 16 mm. camera (see Fig 2, A) with a standard lens mounting can be used for this work. The standard or

"screw thread" mount gives the firmest possible support to the lens and makes the insertion of the adapter (or extension ring) between camera and lens a simple matter. This extension ring will be described later. A direct focusing device is a great convenience, especially if it focuses directly through the taking lens; however, this is more important for preliminary experimental work, where a constant check on the focus and size of the field is necessary. We have our shutter so adjusted that when the camera is stopped the shutter is open. Then, by opening the direct focusing window on the side of the camera we can see the exact image just as it is recording on the film.

The weight of the camera is also a consideration. The photographer holds the camera in such a way that the weight is all on the left hand and in a rather strained position. A heavy camera is very tiring and causes the camera man's hand to tremble. Our camera has a light "bakelite" case and so far as we know is the lightest of the standard 16 mm. cameras using 100-foot rolls.

We prefer a two-inch lens to all others. For color or slow motion black and white photographs it is opened to F 1.5. For black and white photographs at normal speed the diaphragm is adjusted to the Wesson speed of the film emulsion we are using. The lens (see Fig. 2, C), however, is not attached directly to the lens mount, but is moved forward from its usual position by means of a collar or adapter band (see Fig. 2, B). This band is threaded on each end. It is screwed directly into the camera mount and the lens in turn is then attached by the screw thread to the distal end of the adapter. The band is 0.6 cm. wide.

With this lens mount arrangement and the lens open to F 1.5 there results a sharp focus at $11\frac{1}{2}$ inches (29.2 cm.) and a usable focus between 11 and $12\frac{1}{2}$ inches (27.9 and 31.7 cm.), measured from the front of the lens. To maintain the camera at this exact distance from the vocal cords and to exactly align the laryngoscope and the camera, we have built a hollow, tapered metal tube (see Figs. 3 and 4), which at one end is threaded over the lens, and at the other end is exactly the diameter of the proximal orifice of the laryngo-

scope. Bearing in mind that the direct laryngoscope is $7\frac{1}{4}$ inches (18.3 cm.) long, and that we desire to maintain the camera at a distance of $11\frac{1}{2}$ inches (29.2 cm.) from the vocal cords, it is obvious that this tube must represent the difference between these two lengths, or $4\frac{1}{4}$ inches (10.7 cm.) Our tube is telescopic over a distance of about an inch to allow for variations, such as an extremely deep larynx. In this case we then foreshorten the tube. This telescopic arrangement also permits photographing structures which are more proximal, such as the arytenoids. Ordinarily, however, we maintain a fixed tube length of $4\frac{1}{4}$ inches (10.7 cm.) and vary



Fig. 3. The hollow, tapered metal tube (side view). (A) Screw threads for adaptation into front of lens. (B) Threaded sleeves to adjust length of tube. (C) Guide cuff overriding barrel of laryngoscope and fitting into angle between barrel and shaft. (D) Rigid fixation prongs inserting into laryngoscopic channels. (E) Curved arms to fit under laryngoscopic light carriers.

it only rarely. This fixed length usually suffices to bring all laryngeal structures into fairly critical focus.

The distal end of the metal tube (see Figs. 3 and 4) which fits onto the laryngoscope requires special attention. Its superior aspect is lipped or cuffed (see Figs. 3, C, and 4, C) to fit over and rest upon the barrel of the laryngoscope, snugly against the shaft. Two tapered fixation prongs (see Figs. 3, D, and 4, D) project from the tube to insert into corresponding channels which have been soldered or welded to laryngoscope on each side of the shaft (see Fig. 1, D). This arrangement produces great stability and prevents all lateral or vertical movement between the tapered tube and the laryngoscope. It is, therefore, actually self-aligning. The

camera, adapter band, tapered tube and laryngoscope are shown attached to one another in Fig. 5.

In arranging and building these various pieces of equipment it is not wise to follow exactly the dimensions given above, but rather to construct each item bearing in mind the single vital point that an object placed at the end of the laryngoscope must be in focus upon the film and well centered. For instance, our own equipment will not produce good focus

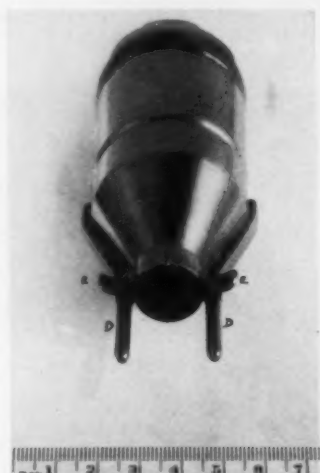


Fig. 4. Hollow, tapered metal tube (front view). (C) Guide cuff. (D) Rigid fixation prongs. (E) Curved arms.

with a certain camera because the lens mount screws more deeply into the camera than in our own. When using this second camera we, therefore, find it essential to appreciably widen the adapter band. Such variations as these cannot be avoided, and each adapter band and telescopic tube must be made for a particular camera and a particular laryngoscope, centering one with the other and rearranging the length of each to establish proper focus and alignment. These factors can be tested during the course of construction by sighting or photographing printed matter placed at the end of the laryngoscope in a position comparable to that of the vocal cords.

For color pictures, Eastman's Type A Kodachrome is entirely satisfactory. For black and white, we use both negative and positive reversal films, depending upon circumstances. If the finished picture is to contain both color and black-and-white shots it is better to use a reversal film (such as Eastman's XX) because the emulsion of both the black-and-white and color will be on the same side of the finished film. Titles can then be made to correspond and neither titles nor subject matter will project out of focus. But if the work is to be all in black and white, then a negative film is much to be preferred. Fine red sensitive emulsions are available

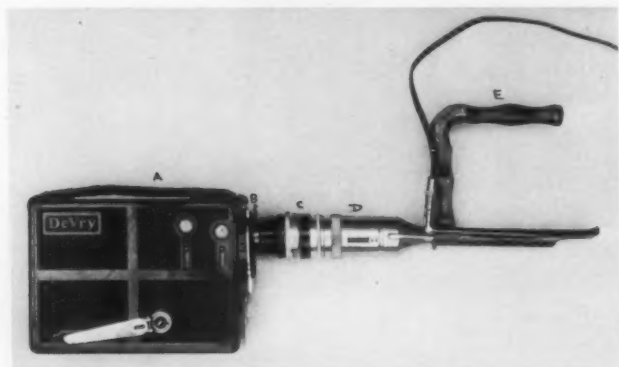


Fig. 5. Portions of apparatus adapted to each other. (A) Camera. (B) Adapter band. (C) Lens. (D) Tapered tube. (E) Laryngoscope.

in the fastest speeds. Editing can be done before the final printing so the finished work is unspliced, and it is also possible to correct for exposure and contrast in printing the positive.

Camera speed varies, but about 12 to 16 frames is satisfactory for color pictures, and in black and white up to 128 frames. The adapter and extension tube are threaded into position and the lens warmed by wrapping a hot water bottle around it for a few minutes prior to taking photographs. This is very important since it prevents fogging of the lens by the patient's breath.

The laryngoscope is inserted by the surgeon and the larynx exposed. At a given signal the photographer, standing behind

and to the right of the surgeon, advances, bringing the camera to the laryngoscope. The right hand of the surgeon grasps the extension tube, guiding the prongs into their laryngoscopic receptors, and through a co-operative movement by both men, the camera is brought home into place and the photograph taken. The entire procedure requires but a few moments, and with practice such results as demonstrated in Fig. 6 may be expected.

By the use of this method we have been able to demonstrate photographically many physiologic phenomena, including respiratory movements of the larynx, the lubricating mechan-

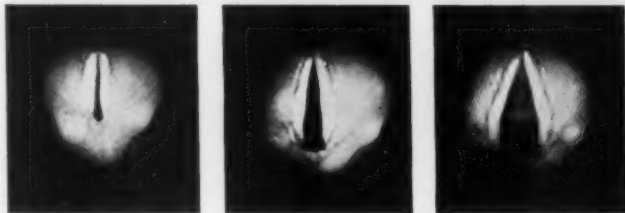


Fig. 6. Single frames reproduced from Kodachrome Laryngeal Motion Pictures.

ism, the appearance of the larynx in the production of tones of varying pitch and the whispered mechanism, the sphincter mechanism in cough, swallowing and thoracic fixation and pathological changes of varying types. Some of these are in slow motion and demonstrate the details of movement with gratifying clarity.

The apparatus used has been partially designed and in the greater part has been made for us by the L. C. Isom Instrument Co., 1416 North Vermont avenue, Los Angeles.

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1917 Wilshire Boulevard.

(a) — PRIMARY CARCINOMA OF THE MIDDLE EAR
AND MASTOID.*

(b) — THE ENDAURAL APPROACH TO THE MASTOID.

DR. CLARENCE H. SMITH, New York.

According to Robinson, in 212,000 ear cases treated at the Manhattan Eye, Ear and Throat Hospital over a period of 20 years, new growths of the ear occurred in the ratio of one in 4,000 cases. In 24 of these patients referred to him for radiation, the external ear and auditory canal were primarily involved in 19; in five cases the tumor arose in the middle ear, one of which was metastatic.

You will see from these figures that primary carcinoma of the middle ear is rare. The growth is invariably of the squamous cell type, in spite of the fact that the tympanic cavity is lined with columnar epithelial cells. The question arises as to how a squamous cell carcinoma can spring from columnar cells.

The sustained irritation of chronic middle ear suppuration can change columnar into squamous epithelium. Chronic suppuration is the leading factor in producing epithelial alteration.

Then again, squamous epithelium may invade the middle ear as in secondary cholesteatoma. Manasse states that this ingrown epithelium has a tendency to pile up and predispose to a malignant degeneration.

Most of the primary cancers of the middle ear are preceded by chronic middle ear suppuration. But how about the cases which have no suppurative ear condition and which show carcinoma in the middle ear with an intact drum?

Berendes reports a case of this kind which was carefully observed in the disease and in which, undoubtedly, the process arose in the middle ear. Berendes, in theorizing as to how a squamous cell growth could develop from columnar cells, calls attention to the fact that malignant degeneration has a pre-

*Read at the meeting of the New York Academy of Medicine, Section on Otolaryngology, May 3, 1939.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 15, 1939.

dilection for sites in which two epithelial surfaces of different types come in contact with each other. He shows that the drum is covered on the inside by the endoderm of the middle ear and on the outside by the ectoderm of the external meatus. Here are two epithelial layers of different types in close contact, and atypical growths may develop.

Goerck's theory is that an isolated squamous epithelial rest may take on new growth and develop into an epidermal carcinoma, as epithelial rests do in the gall bladder and in the body of the uterus, and become starting points of carcinoma.

In my experience, I have recognized and treated three cases of primary carcinoma of the middle ear.

The first of these cases was seen over 25 years ago. On account of the length of time which has elapsed since then, the records are no longer available. The patient was a middle-aged woman who had had chronic purulent otitis media, and the growth started in the middle ear. I did a radical mastoid operation on her, and she was later treated with radium at the Memorial Hospital. She had an extension of the lesion into the brain and succumbed.

The second case, K. G., male, age 58 years, was first seen at the Morrisania City Hospital on June 17, 1936. He complained of pain and deafness in the right ear of two months' duration. Coincident with these symptoms, he had a facial paralysis and a painful preauricular swelling. There was a serosanguineous discharge from the ear. There was an irregularly-shaped, friable, pearly-white fungating mass in the external auditory canal. This mass protruded from the tympanic cavity. Biopsy showed a squamous cell epithelioma. A radical mastoid operation, on June 30, revealed the middle ear filled with cancerous tissue. The walls of the tympanum were deeply eroded by the disease. The dura was found exposed and it transpired that it was invaded by the growth. He was intensively treated with X-ray and radium. He died on Aug. 12.

The third case is that of the patient presented this evening. His history is as follows:

On Jan. 1, 1938, James C., age 61 years, consulted Dr. F. M. Bannon, of Stamford, Conn., on account of pain in the right ear, accompanied by discharge.

Two years previously, he said he had a cold in the right ear, followed by fullness and noises, together with deafness. He was treated at the Manhattan Eye, Ear and Throat Hospital by inflation. He stated that the fullness and the noises in the right ear persisted through these two years.

Examination showed, in the right external auditory canal, a mass which bled easily on manipulation and which seemed to originate from the middle ear. There was a watery, yellowish-red, foul-smelling discharge. He was totally deaf in this ear. The facial nerve was intact.



Fig. 1.

The mass was irregular in shape and hard and gritty to the touch. A biopsy was performed. The specimen was examined by a pathologist, who reported the presence of "a squamous epithelioma, probably from the canal wall." I show you now a microphotograph of the tissue. This was examined by Dr. Fred Stewart, of the Memorial Hospital, who confirmed the diagnosis as epidermoid carcinoma, Grade 1.

I saw the patient on Jan. 6, 1938, and found a growth filling the right auditory canal. I referred him to Dr. G. Allen Robinson, who reports as follows:

"Radiation treatment consisted of the implantation of five gold radon implants, 1.0 mc. each, giving a total dosage of 666 mc. hours. Three of the implants were used on Jan. 7, 1938, one implant on Jan. 24, 1938, and one implant on March 2, 1938. In addition to the interstitial radium treatment, deep Roentgen ray therapy was employed. A total of 4,400 'r' units were administered to the external auditory canal and mastoid area. The following are the radiation factors:



Fig. 2.

"Target skin distance, 50 cm.; thoreaus filter; 200 K.V.P.; 22 treatments, 200 'r' per treatment; circular field of 7 cm. diameter.

"One-half of the radiation treatment was given preoperatively, and the other half of the treatment postoperatively."

He was admitted to the Bronx Eye and Ear Infirmary, April 13, 1938. I performed a radical mastoid operation on him on April 14. A considerable amount of necrotic and granulation tissue was found in the middle ear. There was no tumor mass. The bony walls were unusually necrotic. The facial ridge was markedly softened. The external auditory canal wall was free from involvement. The diseased tissue was removed and, macroscopically, a healthy bony cavity was

attained. The next morning, facial paralysis developed. He left the hospital one week later.

His ear was completely dry nine weeks after the operation. The facial paralysis at that time was improving, and shortly afterwards it cleared up completely.

These cases of primary carcinoma of the middle ear are reported to place on record three more instances of a rare otological condition. The third case is reported in detail to illustrate the value of modern radiation therapy combined with surgical extirpation.



Fig. 3.

(b) — THE ENDAURAL APPROACH TO THE MASTOID.

The endaural approach to the mastoid was first introduced by Kessel¹ in 1885, and was often used by Hoffmann between 1892 and 1894. It has since had several advocates and it has been the subject of intermittent waves of popularity. It has not escaped condemnation by some writers. Much of this adverse criticism was doubtless caused by faulty technique.

Lempert, of New York, has been the leading exponent in America of this procedure. He has perfected steps which, when properly followed, insure adequate exposure of the cortex over the mastoid antrum and the surrounding bone.

I have not attempted the simple mastoid operation through this opening. Howarth and Bateman,² in a recent report of their experience in 30 cases (20 being affected with acute mastoiditis), are enthusiastic about the endaural technique.

In the past year, I have used the endaural approach in nine modified radical and in eight antrotympanic radical mastoid operations. I like this approach in these middle ear operations because it affords an excellent exposure of the tympanum and of the attic structures. The operator has the advantage of a direct vertical view of the operative field in the endaural route, instead of an oblique view given through the retro-

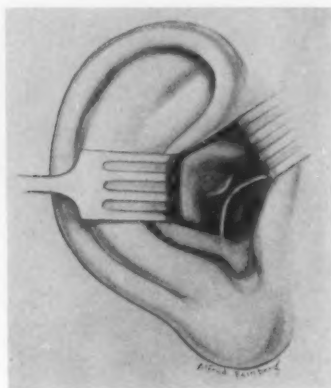


Fig. 4.

auricular incision. This improved visibility is valuable in preventing subluxation of the ossicles and in guarding the drum in the modified radical operation.

Lempert's³ technique consists in making a triangular window in the posterosuperior meatal wall. A complicated description of the incisions outlining the area makes this step perplexing. It will simplify it if one thinks of it in this way: the base of the triangle is formed by an incision running from the lower part of the meatal wall directly in front of the concha, through the soft tissues around the canal, to terminate in the upper angle of the space between the tragus and the helix. The apex of the triangle is a point in the postero-

superior meatal wall at the junction of the osseous and membranous portions. The two sides of the triangle are formed by joining this apex with the upper end of the first incision and by joining the apex with the lower end of the first incision. These incisions do not go through muscle or cartilage. Before making the triangle, it is advisable to inject the canal wall with a 1-10,000 solution of adrenalin chloride. This gives good hemostasis, and one rarely has to use a hemostat.

The outlined triangular area is elevated from the bone and is discarded. The periosteum over the mastoid process is undermined. With retractors, distensibility to any desired

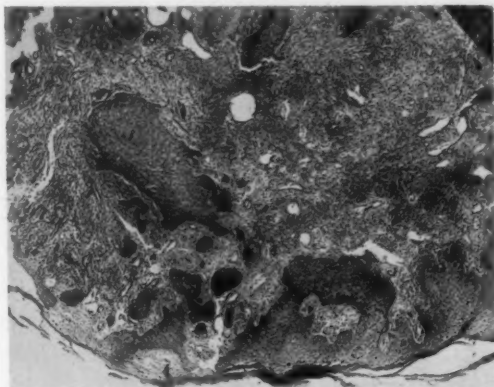


Fig. 5.

extent is easy. During the mastoid operation, the rest of the membranous canal is carefully preserved. When the modified or the radical mastoid operation is completed, the membranous canal is divided vertically to help in covering the denuded bone. The upper arm of the divided membranous canal wall is reflected over the remaining portion of the superior bony canal wall, the lower arm is folded over the facial ridge.

Epidermization quickly spreads from the margins of these flaps, and in about three weeks' time the antral area and the epitympanic space are completely epithelialized. I have used a skin graft in the middle ear in the radical mastoid operations. Using the membranous canal wall in this way obviates

the necessity of making one of the usual meatal flaps, with the risk of an occasional perichondritis.

The upper edges of the excised triangular area fall together. There is no loss of tissue except the removed bone, because no muscle or cartilage has been cut. The postoperative cosmetic appearance is excellent. On healing, the operated ear looks the same as its fellow.

Since adopting this approach, I have used the bone drill and burr, instead of the gouge and chisel. This change saves labor and time in working on the sclerotic bone usually found in these chronic suppurative cases.

To illustrate the postoperative results after using this technique, I present this evening two of the patients on whom I have performed the modified radical mastoid operation. One of these was my first endaural attempt, on June 15, 1938; the other was operated on March 8, 1939. The first patient had had purulent otitis media for 12 years, with a large perforation in the posterior inferior quadrant. His ear was dry nine weeks after operation. Since then, his ear has remained dry and his perforation has closed. His hearing is excellent.

The second patient had had a purulent otitis media for 30 years. There was a fair sized perforation in the region of the umbo. His ear was dry five weeks after operation. The perforation has closed. His hearing is excellent.

Altogether, I find the endaural approach in these operations is a distinct aid on account of the better and more direct view of the operative field. This visibility is particularly valuable in the modified operation, in which the integrity of the ossicular chain and of the drum is all-important in the conservation of hearing. The use of the drill and burr is another benefit as an aid in simplifying what is often a laborious procedure.

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140 East 54th Street.

PUPILLARY REACTIONS IN AFFECTIONS OF THE EAR.

DR. J. BERBERICH, London.

There are numerous causes for differences in the pupils in man; however, in this following article we shall only be concerned with those differences in the pupils that can be traced to the ear. Years ago, Schurygin, Udvarhelyi and Cemach pointed out that constriction takes place in the pupil on the same side as that which has been stimulated by sound. Stimulation of the vestibulum may also produce a change in the pupils, although, according to Wodak, only a dilatation. In practice these reactions have never gained much significance.

Isolated observations of differences in the pupils in diseases of the middle ear have already been made; the most detailed investigation of this kind was undertaken by Spiegel. He discovered among 34 patients with diseases of the middle ear, only in two instances a miosis, and in one an enophthalmos on the side of the diseased ear. These conditions of the eye seemed to be of rare occurrence, as judged by our purely clinical experience, so that mainly for this reason we investigated this problem again.

It is only when working in collaboration with an experienced ophthalmologist on such a question that one realizes how many cases among the in- and outpatients must be removed, since other causes for differences in the pupils might also have to be taken into account in these instances. Thus, cases of anisometropy, of sluggish pupils, of syphilis, of apical tuberculosis, of thyroid enlargement, of neck and throat injuries, etc., have to be eliminated.

To quote only a few examples from the cases that we eliminated:

Patient V. Gr.: On the right ear a radical operation; on the same side, ptosis, miosis, enophthalmos. Since, however, a cirrhotic process of the lungs was also present, as a precautionary measure we decided against using this case, or the following case: Patient K.: The pupil on the side of the radically operated ear was markedly smaller; nevertheless, it was discovered that the patient had suffered from a disease of the cornea, including the aqueous humor, this rendering him unsuitable for exact research work.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 26, 1939.

This strict selection of patients was made possible by the assistance of my colleague, Metzger, an eye specialist, who so kindly offered his services in supervising the examinations and checking the tests.

The examination of the patients was made in a semidark room, using Haabs' pupillometer. Simultaneously with the size of the pupils, the separation of the eyelids was also measured by Wessely's method with the aid of the keratometer; the position of the eyeball was registered exactly, and particular attention was paid to any sign of heterochromy and to iridescence, which is one of the symptoms of Horner's syndrome (Metzger). The tests were repeated on the patients on the same day and at greater intervals, and only constant results were used. In addition, we made use of the pharmacological test, as suggested by Spiegel, and it proved most reliable to inject cocaine into the conjunctival sac, as recommended by Levinsohn, Heine and Minor. After repeated measurement of the pupil on both sides, one drop of cocaine hydrochloricum was introduced into the conjunctival sac, and at intervals of five, 10, 15 and 20 minutes the size of the pupils was measured and compared. In the majority of cases the adrenalin test for ascertaining the differences of the sympathetic innervation of the pupils was also taken into consideration. After measuring the size of the pupils, two drops of a solution of 0.1 per cent adrenalin was instilled into the conjunctival sac. Fifteen minutes later, the first reading was taken, and simultaneously another instillation was made, 15 minutes elapsing before the second reading was taken. Afterwards, instead of synthetic adrenalin, the original extract of the suprarenal gland was used.

In three cases, following the method of Spiegel, we applied a strip of gauze, soaked in 20 per cent cocaine, to the cavity of the radically operated ear in order to secure an inhibition of the sympathetic nervous action. In these cases no change in the pupil was noticed. In two further cases we instilled the same solution through a perforation of the tympanum into the middle ear but could observe no change in the pupil. Spiegel also notes that the results of these tests are not constant.

After eliminating every doubtful case, we examined, in all, 27 patients operated on one side only for diseases of the middle ear. Of these, nine cases showed a difference in the pupils

of over 0.5 mm.; nine cases showed a difference in the pupils of from a trace to 0.5 mm.; nine cases showed no change whatsoever.

In the first group the pupil on the side of the diseased ear was always more than 0.5 mm. narrower than on the healthy side. In seven cases a narrowing of the palpebral fissure was observed. In the remaining two cases, some dilatation was present, most likely due to a weakness of the facial nerve.

In eight out of the nine above-mentioned cases, the cocaine test proved to be a weaker stimulus of the dilator pupillae; *i.e.*, after the instillation of the cocaine, which augments the sympathetic response, the pupil on the side of the diseased ear remained narrower than on the healthy side.

The adrenalin test could only be made in seven cases in this group: In four cases the result was positive, in the sense of an increased response by the eye, as is characteristic of post-ganglionic injuries; in the other cases the adrenalin test was not typical, nor, indeed, did it prove to be as reliable in our examinations as the cocaine test.

In the second group only two cases showed symptoms of sympathetic paralysis; six cases showed even hypersensitivity of the dilator pupillae towards cocaine and adrenalin.

In the third group, with pupils of equal size, the pharmacological test showed in two cases only a hyposensitiveness of the pupils.

Numerous interesting details of the examination will be found in the exhaustive account of my co-worker, Miss R. Weyl.

From these examinations it is, therefore, clear that a very large number of patients with diseases of the ear, or with operated ears, suffer from injuries to the sympathetic nerve near the middle ear. In agreement with the investigations of Spiegel and well known experiments on cats and rabbits, our experiments show definitely that in cases of radical operations in the ear, or of destructive processes in the middle ear, an injury may result to the nerve of the dilator pupillae as it traverses the middle ear. This is manifested in a dilatation of the pupil of the affected side, accompanied by simultaneous weakening of the tarsal muscle (ptosis sympathica); we have never observed complete paralysis of the sympathetic. Consequently, we must assume that the anatomical relations in

man are similar to those in the rabbit, in which, also, after a radical clearance of the middle ear not all the fibres of the sympathetic are destroyed, whereas in the cat the same interference results in complete denervation. The reason that all operated cases do not show a similar disturbance of the dilator fibres, that they do not all show a change in the pupil, can only be explained by the fact that, according to anatomical dissections, the course of the fine nerve twigs in the middle ear, and particularly on the sulcated medial wall of the tympanic cavity, is subject to great individual variation. Besides these individual variations, it must also be assumed that in operations of the middle ear, not merely a part of the sympathetic fibres is destroyed but that a part also is injured or irritated; this is proven by the cocaine tests, which in a large number of cases resulted in a hypersensitiveness of the dilator. The changes in moist and dry cavities of radical operations and various other interesting details need not be discussed here.

SUMMARY.

Diseases of the middle ear and radical operations of the middle ear result relatively often in disturbances of the sympathetic nerve with differences in the pupils.

Out of 27 cases examined, nine showed definite changes in the pupils, in the sense of a weakness of the sympathetic nerve on the operated side. In nine further cases, constant changes in the pupils were present which were less than 0.5 mm. but were unmeasurable; of these, only three showed symptoms of a paralyzed sympathetic, as compared with six cases of hypersensitiveness. Nine further cases had equal pupils.

The clinical results prove that with ear operations, on account of anatomical variations, only a partial disturbance of the sympathetic fibres running along the medial tympanic wall is caused, and that the intact fibres may be irritated by any existing inflammation. The pharmacological tests show the same result.

Considering the relative frequency of difference in the pupils after diseases of the middle ear, it is essential in cases of pupillary disturbances also to pay attention to the condition of the middle ear.

11 Lyndhurst Road.

ATYPICAL MASTOIDITIS — ADULT TYPE. CASE REPORTS.*†

DR. J. DUDLEY SINGLETON, Dallas.

In taking up the discussion of this subject, the term mastoiditis will first be defined as an infection of the mucous membrane, of some or all of the mastoid cells, with or without bone destruction and thrombophlebitis, usually associated with infection and discharge from the middle ear, presenting local manifestations of inflammation over the mastoid, and systemic signs and symptoms that can be definitely attributed to the infection.

Any case presenting unusual abnormalities may be spoken of as atypical; however, in this article the term will be used to designate those cases, occurring chiefly in adults, presenting little or no apparent infection of the middle ear, with almost a total absence of local and general signs and symptoms of the diseases — frequently referred to as primary mastoiditis. Ersner,¹ in speaking of this condition, describes it as "a pathological picture superimposed upon a pathological condition," and calls it "the anarchist of mastoid disease." The author² reported one such case, in 1931, while at the Graduate Hospital as Resident Otolaryngologist, and has operated upon two others; one in Philadelphia, and one in Dallas.

The following cases illustrate this type of the disease:

Case 1: Patient, V. F., female, colored, age 50 years, married, came to the Graduate Hospital, of Philadelphia, Dec. 11, 1931, complaining of pain and swelling behind the right ear, swelling of the upper portion of the right side of the face, and discharge from the right external auditory canal.

History: The family and past histories were irrelevant. The present complaint began about two months before the patient came to the hospital. At the onset, she had a slight cold that lasted for two or three days. About three days following the onset of the cold, she felt a pain in the right ear and behind the ear. The pain was sharp and shooting in character and worse at night. It radiated to the right temporal, parietal and occipital regions of the head and lasted for about three weeks. It then changed to become a constant, dull, aching, boring pain

*Read before the Texas Ophthalmological and Oto-Laryngological Society, San Antonio, Dec. 9, 1933.

†From the Department of Otolaryngology, Baylor Medical College, Dallas.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 14, 1933.

confined to the right mastoid region. About six weeks from the onset of the symptoms, the patient noticed some swelling behind the involved ear. Two or three days after the appearance of the swelling, a thick, yellow discharge was observed coming from the right external canal, which was continuous thereafter. On Dec. 10, 1931, two months after the initial symptoms, the right side of the patient's face became swollen, and as a result of this swelling she decided to seek medical aid.

On examination, the tissues behind the right ear were edematous and there was some fluctuation. There was considerable swelling over the upper portion of the right side of the face. The external auditory canal on the right side was filled with thick, yellow pus. On making firm pressure over the right antrum, about 2 or 3 cc. of pus flowed from the canal. On the posterior canal wall, about 1 cm. external to the drum membrane, a small, cone-shaped, papillary-like projection was seen protruding into the canal. Pressure was again made over the antrum and pus was seen to flow from the free end of this projection. The tympanic membrane was slightly reddened in its upper portion, showed some loss of lustre, but there was no bulging or perforation.

The patient was admitted to the hospital on the service of Dr. George M. Coates for further study and treatment. On admission, her temperature was 98.6°, pulse 84, respirations 20. The blood count showed 5,780,000 red cells, 75 per cent hemoglobin, 6,700 leukocytes, 62 per cent polys., 25 per cent lymphs., 2 per cent eosinophils and 1 per cent monocytes. The urine was normal.

X-ray pictures of the mastoids showed the left clear. The right was reported as follows: "Subacute suppurative mastoiditis with abscess about 2 cm. in diameter present just below the knee of the sinus and extending down toward the tip." As a result of the X-ray and clinical findings, an immediate mastoidectomy was decided upon. The operation was done Dec. 11, 1931, at 9:15 P.M., under an ether anesthetic.

Operative Findings: The usual mastoid incision was made, and the overlying soft tissue were edematous and thickened. A subperiosteal abscess with a perforation of the cortex over the antrum was found. The mastoid cells were broken down and the cavity was filled with pus and granulations. There was a fistula through the posterior bony canal wall connecting the mastoid cavity with the external auditory canal. The lateral sinus was found exposed over a large area, and was covered with granulations, but it did not appear to be thrombosed. The mastoid cavity was thoroughly cleansed to healthy, hard bone, and most of the posterior bony canal wall was removed. The wound was packed lightly with one-half inch iodoform gauze and closed in its upper two-thirds. Recovery was rapid and uneventful.

Case 2: Patient, H. T., male, white, age 17 years, single, came to the Baylor Hospital Clinic, March 7, 1934, complaining of a tender swelling about 3 or 4 cm. back of the left ear.

History: The family and past histories were negative. The present complaint began about one month before the patient came to the clinic, with a severe head cold and sore throat. About one week after the onset of the cold, both ears became very painful, and there was swelling and redness back of the left ear. The swelling and redness disappeared in about 10 days or two weeks, and the patient felt much better. About four or five days before the patient came in for medical aid, he had a severe aching pain back of the left ear. A day or two later, swelling appeared well back of the ear and gradually increased. There was never any drainage from either ear. General systemic symptoms were not complained of after the first few days of his illness.

On examination, a fairly large tender fluctuant tumor mass about 3 or 4 cm. in diameter was seen and felt under the scalp well back of the left ear. The postauricular sulcus was not obliterated. There was no sagging of the posterior superior bony canal wall. The drum membrane showed some loss of lustre. There was no redness, bulging or perforation. The right ear was normal in appearance. Examination of the nose showed no discharge or obstruction. The tonsils were hypertrophied and chronically inflamed. The pharynx was red and somewhat granular.

The patient was admitted to Baylor Hospital, March 7, 1934, for a more complete study. On admission, his temperature was 100°, pulse 118, respirations 20. The blood count showed 11,300 leukocytes, 76 per cent polys., 20 per cent small lymphs., 3 per cent large mononuclears and 1 per cent eosinophils. For some reason a red count was not made. The urine showed a trace of albumin.

X-ray pictures of the mastoids were reported as follows: "Films of both mastoids show these structures to be quite large and made up of rather small, thin wall cells. There is some haziness made out in both mastoids, and this process seems to be most marked in the anterior portion of the left mastoid. The posterior cell structure is somewhat irregular on both sides. Opinion: Slight changes in both mastoids suggesting the presence of an old infection most marked on the left side."

As a result of the clinical and X-ray findings, a mastoidectomy was decided on. The patient was operated upon, March 8, 1934, at 1:15 P.M., under an ether anesthetic.

Operative Findings: The usual mastoid incision was made and from it a T-shaped incision was extended back into the fluctuant mass. A large subperiosteal abscess was found over the upper posterior portion of the mastoid cavity. In this same location there was a small round perforation of the cortex, about 5 or 6 mm. in diameter. The mastoid cells were completely filled with edematous mucous membrane and a small amount of pus. There was a large exposure of the lateral sinus. The sinus was covered with granulation tissue. The entire mastoid cavity was thoroughly cleansed to hard, healthy bone. About one-third of the posterior bony canal wall was removed. The wound was flushed out with normal saline solution, packed lightly with one-half inch iodoform gauze and closed with dermal in the upper two-thirds. Recovery was rapid and uneventful. At no time after the operation did the temperature reach 100° F. The patient was discharged from the hospital, March 15, 1934, with a normal temperature and a very slight drainage. Cultures from the mastoid showed a streptococcus infection. One week later, the wound was entirely dry.

A number of men have written on the subject of so-called primary mastoiditis and reported cases illustrating this type of the disease. Hetrick,³ of New York, wrote a very excellent paper on this subject in 1928. He reported four cases and reviewed 48, including his own. All were operated upon and recovered. Hemstead,⁴ in 1926, reviewed the literature and reported six cases. In one of his cases the mastoid infection apparently was secondary to a furuncle of the external canal. All made uneventful recoveries after being operated upon. MaCuen Smith,⁵ in 1927, reported four cases of atyp-

ical mastoiditis; however, only two of these cases did not have drainage from the middle ear before the operation. In one of his cases the mastoid infection evidently was secondary to a furunculosis of the external canal. His patients were all operated upon and recovered. In 1925, Kopetzky and Almour⁶ reported three cases and summarized them as follows:

"These three cases present the following factors in common:

"1. An absence of any aural discharge prior to the present.

"2. The finding of a normal membrana tympani at all stages of the disease.

"3. The failure of the disease to produce even a moderate local or constitutional reaction.

"4. The finding of a subperiosteal abscess and perforated cortex.

"5. An erosion of the inner table, with an involvement of either the sinus or dura."

All recovered from the operation. But one, a child, age 8 years, who gave a history of repeated attacks of acute nephritis for six years, developed acute glomerular nephritis and died in uremic coma six weeks after the operation. MacKenzie,⁷ in 1929, reported a case. The patient was operated upon and recovered. Mollison,⁸ in 1921, reported two cases. Both had lateral sinus thrombosis. Both were operated upon and recovered. In 1917, Long⁹ reported a case complicated with perisinus abscess, phlebitis and streptococcemia. The patient was operated upon and recovered. In 1907, Bennett¹⁰ reported a case of mastoiditis without symptoms, complicated by meningitis, which terminated in death.

DISCUSSION.

Patients suffering from this type of infection present a number of more or less common interesting features for consideration. In reviewing the reported cases, one finds that it occurs most often in adults, but it does occur in infants and children. In most cases there is a history of a preceding upper respiratory infection with pain in the ear at the time

or within a day or two. The pain may be mild or severe. It usually disappears in a few days. Occasionally it is persistent. Following the pain, there is often a quiescent period, with almost a total absence of symptoms and complaints, lasting from a few days to several months, or longer in some cases. General systemic symptoms, if present, are mild and may pass unnoticed by the patient. The mastoid disease may sometimes be secondary to a localized infection about the ear, such as a furuncle. Hemstead and MaCuen Smith have both reported such cases. Physical signs are few. Tenderness over the mastoid may be present, but is usually very mild. There may be a slight thickening of the periosteum over the affected mastoid. If present, it will have a smooth, soft feel, as compared with the hard, rough bone of the opposite side. Sagging of the superior posterior bony canal wall is usually present. It is the most constant and often the only local sign indicating the presence of mastoid infection. It is said to be pathognomonic of mastoid disease in the absence of local external infection. MacKenzie, in his discussion of Heterick's paper on this subject in 1928, stated that "mastoiditis may exist without the history of middle ear suppuration and without perforation of the drumhead; but hardly without swelling of the periosteum of the superior posterior wall of the osseous canal." He has, however, since reported a case in which the above sign and all other signs of mastoid disease were absent at the time of his examination. Occasionally the temperature will show a slight elevation, but this is not constant. Swelling of the soft tissues about the affected ear frequently occurs late in the progress of the disease. It usually develops suddenly and with little or no pain, and is most likely to be postauricular. It may occur in the zygomatic region, well in front of the ear, or in the neck as a Bezold's abscess. X-ray pictures of the mastoids will almost invariably show clouding and evidence of bone destruction.

The diagnosis of this condition is rarely made early. This is explained in large part by the fact that these patients seldom seek medical aid until alarmed by the sudden appearance of swelling and pain about the ear. If one is to make an early diagnosis, the possibility of this type of infection must be kept in mind and patients presenting themselves for treatment with a history of an upper respiratory infection,

followed by pain, mild or severe, constant or intermittent, in the region of the ear should be given a thorough otologic examination, including X-ray pictures of the mastoids. Repeated examinations should be made, if deemed advisable. In the presence of the above history and with sagging of the superior posterior canal wall in its bony portion, a diagnosis of suppurative mastoiditis can be made if local external infection is eliminated. As an aid in the diagnosis, Hetrick mentions the "Weber-Schwabach Paradox of MacKenzie," where the Weber lateralizes to the diseased ear from the vertex, but gives a shortened Schwabach when the instrument is placed upon the diseased mastoid. Good X-ray pictures usually give the most definite and deciding information in these cases.

In the differential diagnosis one must exclude other conditions causing pain or distortion of the tissues about the ears. The most common of these are:

1. Infections of the external auditory canals.
2. Suppurative postauricular adenitis.
3. Referred pain from the nose, mouth or throat giving mastoidalgia.

The treatment of these cases is always surgical. It should consist of complete exenteration of all diseased and necrotic bone and all available cells in the mastoid process, followed by good postoperative care.

The pathology as seen at the time of operation usually consists of extensive bone necrosis and cell wall destruction, with pus and granulations filling the mastoid cavity. Perforation of the cortex with subperiosteal abscess formation, and erosion of the inner plate with perisinus and extradural abscess formation occur with great frequency, and are often referred to as complications of the disease. The more serious complications of generalized meningitis, brain abscess, lateral sinus thrombosis and septicemia rarely occur.

Cultures from the mastoids show a variety of organisms, with streptococcus and pneumococcus type III predominating in the order named.

Prognosis is excellent in the absence of the more serious complications. These patients usually make rapid and complete recoveries, barring some slight hearing defect.

SUMMARY.

Atypical mastoiditis, the so-called primary mastoiditis, occurs chiefly in adults, but may occur at any age. It presents little or no evidence of infection in the middle ear and gives very few local or general signs and symptoms of the disease. An early diagnosis of the condition is rare. Important aids in making an early diagnosis are:

1. A careful history of the case.
2. Evidence of periostitis as shown by sagging of the superior-posterior bony canal wall.
3. Good X-ray pictures of the mastoids.

Extensive bone destruction with perforation of the cortex and subperiosteal abscess formation occurs often. Erosion of the inner plate with exposure of the lateral sinus and dura is also common. Serious and fatal complications are rare. Streptococcus and pneumococcus type III are the organisms most frequently found, and occur in the order named.

CONCLUSIONS.

1. Atypical mastoiditis, the so-called primary mastoiditis, occurs chiefly in adults.
2. It runs a mild course, with few local or general signs and symptoms of the disease.
3. The presence of edema in the superior-posterior bony canal wall and good X-ray pictures of the mastoids are the most reliable diagnostic aids.
4. Fatal complications are rare.

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1719 Pacific Avenue.

THE CASE OF "SIR" MICHAEL — A "RADICAL MASTOIDECTOMY" WITH UNUSUAL FEATURES.

DR. LOUIS R. EFFLER, Toledo.

Introduction: We shall call him "Sir" Michael! The immediate circumstances of his case were reported by us preliminarily at the regular staff meeting of Mercy Hospital, Toledo, on March 15, 1938. Since then much water has gone over the dam. We have purposely waited to learn the exact final outcome of his case before submitting a complete final report to the literature on the subject.

Diagnosis: The case of "Sir" Michael represents the type that turns a surgeon's few remaining hairs gray with worry. Beginning as a *chronic suppurative mastoiditis* complicated with *cholesteatoma* and "*fistula syndrome*" (which are bad enough in themselves), it ended with an "*hourglass*" mastoid-anomaly and a *far forward-lying lateral sinus* as extra surgical hazards plus a *facial paralysis* (inflammatory) and a severe *carbuncular wound-infection* as postsurgical complications. Worry! Worry! Worry! Despite the multiplicity of hazards and complications, it all ended happily in complete recovery. Yet, who could have foreseen this at the time? Truly, *finis coronat opus!*

Past Clinical History: "Sir" Michael had been under our observation for the better part of 20 years. When first treated by us he was suffering from a bilateral chronic suppurative (hyperplastic) otitis media. Both ears were running furiously with a foul-smelling discharge. The right ear was obviously the worse. These middle-ear abscesses dated back to a scarlet fever attack in childhood. Both were "attic" involvements with perforations in Shrapnell's membrane.

Past Clinical Treatment: After cleaning up an acute suppurative antritis, bilateral, that had flared up the chronic into acute-chronic ear processes, we were ready to go ahead and see what progress we could make with the ears themselves.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, April 10, 1939.

A *left* aural polypectomy, plus a careful curettement of underlying granulation tissue, was successful in yielding a *dry* ear. This ear has remained consistently dry to the present date.

The *right* ear, however, under similar treatment failed to respond to our ministrations. The reason was a *cholesteatoma*. Despite every "dodge" and trick we were able to muster, the ear continued to run. It is true the discharge was much lessened; so much so, in fact, that a greenish crust would form periodically over the perforation. Just as periodically, this crust would necessitate removal because it evidently caused pressure or dammed back secretions sufficiently to produce labyrinthine dizziness. Removal of the crust partly relieved dizziness. Our diagnosis at the time was *chronic mastoiditis with a complicating fistula syndrome, right*.

Interim Observations: Matters went on this way for many years. We had frequently explained the danger of the situation to our patient and, as frequently, suggested the need for surgical interference. His fears, however, overcame his prudence.

Then came the depression! With it, our patient fell into bitter days. Too proud to accept treatment at our hands, for which he figured he was unable to pay, and too proud to explain the situation to us, he sought help at the County Hospital. We lost track of him for a period of two years.

Present Clinical History: Our patient finally presented himself at our office in desperate straits (Sept. 1, 1937). Disheartened, he admitted he could go on no longer. He was so dizzy that he could no longer stand. Attempts at walking caused him to stagger to his right. Palpably, any manual labor was out of the question. Stooping produced an insufferable vertigo.

Present Surgical Treatment: What to do? We performed a *right radical mastoidectomy* on Oct. 7, 1937, in the hope that 1. we might eradicate obviously diseased bone; and 2. we might close over his labyrinthine fistula with healthy granulation and thus stop his dizziness.

Unusual Findings at Operation: The uncapping of the mastoid revealed an anomaly the like of which in severity we had never seen. We had heard of "*hourglass mastoids*" and had even met some mild ones. But, here was the grandfather of all such. Here was one in which the centre of constriction measured hardly more than one-quarter inch across. Those of you who have had more than average mastoid experience may picture the difficulties attending such an operation. We sweated gloriously. Coupled with a densely eburnated bone, a *forward-lying lateral sinus* gave room for scarcely more than the admission of a probe into the aditus ad antrum. Luckily, we were able to locate and to pass a right-angled probe into the antrum from the middle ear side. Leaving this probe *in situ* as a guide, we were able, thus, gradually to hew out the pyramidal wedge of bone that converted the mastoid and the middle ear into one cavity. Without this probe as a guide, we could never have completed the operation successfully. We recommend this unorthodox method to others in similar difficult anomalous cases.

Severe Sequelae of Operation: Imagine our chagrin when, next morning, we visited our patient in the hospital and noted not *one* but *two* most distressing sequelae: 1. A complete right facial paralysis; and 2. one of the most fearful postoperative infections we have ever seen in any locality.

1. *Facial Paralysis:* Naturally, the fear of a surgical facial paralysis is the *bete noir* of every mastoid surgeon. Fairly confident in the face of normal anatomical relations, the surgeon is at times completely helpless in the face of certain mastoid anomalies. Here was an anomaly at the operation, in the shape of a third degree "*hourglass mastoid*," that might conceivably have left the facial nerve anywhere but where it might logically be expected to course. Of only two things were we certain at operation: *a.* that we had taken unusual pains to avoid its possible injury; and *b.* that we had not exposed it to view. In the face of these two facts, we could but pray that the facial paralysis was nonsurgical and, therefore, temporary; and that its cause (instead of being surgical) was probably inflammatory, due to the unusually severe secondary infection that we are about to describe.

2. *Postoperative Carbuncular Infection:* There was nothing unusual in the preoperative appearance of this mastoid-

itis. There were not even any acute, but only chronic, manifestations. It is true a moderate fetid mucopurulent discharge had been observed coming from the middle ear; but, in the face of a low grade temperature (99.5°), this evidence of a chronic involvement had appeared only to be expected. Furthermore, at operation, the mastoid labyrinth was found completely uninvolved. Except for its stony hardness, all pathology seemed to be limited to the mastoid antrum and the attic, including Prussak's space, all of which were found completely filled with granulation tissue, fetid mucopurulent discharge, and profuse cholesteatomatous debris. Nothing unusual, therefore, in the operative findings!

But something decidedly unusual in the postoperative reaction! We would have welcomed pus as something that could be drained. Instead of pus, however, both the lips of the wound and the tissue for a distance of two or three inches from the edges of the wound were swollen to unusual thickness. Palpation expressed no frank pus in any direction. Instead, a stony or wooden hardness was reminiscent of the so-called "ligneous phlegmon" to be seen at times in the tissue of the neck or in Ludwig's angina under the tongue.

These densely indurated tissues were at least two inches thick, very red and very tender. To secure the maximum of drainage, the wound had been spread widely open early and all the stitches removed. After the lapse of two days, the exposed edges of the wound, together with each stitch opening, began to develop a peculiar whitish membrane. Smears taken from the membrane revealed staphylococcus albus. For 21 days, this state of affairs continued. There seemed to be no softening of the densely indurated tissues. They were truly *carbuncular* in character.

Meanwhile, the patient was getting a marked toxic absorption from the wound, as was evidenced by his continuous temperature for three long weeks. Slowly, at the end of this time, the tissues about the wound then began sloughing away. Large cores were picked away *en masse*, exactly as in a clinical carbuncle. It was only when the last of these cores were sloughed off and gentle expression yielded a rich blood flow that healthy granulation became apparent and the wound began slowly to heal.

Postoperative Office Treatment: The patient remained in the hospital for six weeks. It took this length of time for all temperature reaction to subside. The wound, of course, was still far from healed. As a result, further office treatments were continued daily for three more months. Slowly but surely, the depleted mastoid tissues began to regenerate. With proper coaxing, by means of rigorous surgical cleanliness and judicious plastic repair, the wound was completely granulated at the end of this time.

Meanwhile, the facial paralysis continued. So certain were we by this time that the paralysis was inflammatory that we did not bother to make certain electric tests, *viz.*, reaction to degeneration, to prove our point. We felt recovery would be a matter only of time. Accordingly, we outlined a course of daily massage for the flattened muscles and continued to wait in patience.

On April 10, 1938, exactly six months after operation, our patient reported his first ability partially to close his right eye. From this point, week after week, a slight further improvement would become manifested. First a wrinkle or two in the right forehead, next a pursing of the right lips, then a puckering of the right nostril . . . and finally, at the end of one long year, complete regeneration of all facial branches and complete restoration of function to all right facial muscles.

Summary of Final Results: In the face of these severe and unusual complications, the postoperative end-result exceeded our fondest expectations. Exactly one year from date of operation, our patient was able to report:

1. A completely healed mastoid wound with a far better than average cosmetic result.
2. A completely dry middle ear, which, despite one's best efforts, is a result that is often denied a big percentage of radical mastoidectomies.
3. A complete recovery from a severe complicating post-operative facial paralysis.
4. A complete recovery from a postoperative complicating carbuncular wound infection.

5. A complete recovery from staggering and dizziness, both in walking and on stooping.

6. The restoration to society of a useful member in place of a chronic invalid.

After-word: This report would not be complete without a tribute to several fine qualities displayed by "Sir" Michael himself. Despite his severe anteoperative mental and physical disturbances, his severe postoperative shock and, above all, his long-drawn-out painful and discouraging postoperative complications, he showed a brand of *courage* throughout that was highly commendable. His *confidence*, moreover, in the ultimate success of our surgical efforts was enough to buoy us up with new hope and keep us from becoming discouraged when things looked blackest. Lastly, his *gratitude* was genuine. In our present state of social unrest and economic turmoil, gratitude is one of the lost virtues. Our patient, however, was not satisfied merely to express his thanks. He was determined to prove it. What was our surprise one fine day, after he had secured a W.P.A. job, to have him march into our office and *pay us on account a \$5.00 bill from his first \$15.00 pay check!* If he never pays us in full, we are richly rewarded. By this one manly gesture, our more or less lost confidence in human nature has been again largely restored! Also, another foe against State Medicine has been gained. He says so! All praise to "Sir" Michael!

222 Michigan Street.

GOLD PROTHESIS IN DRUM DEFECTS.

DR. SIMON L. RUSKIN, New York.

The value of closing defects of the tympanic membrane was recognized as far back as 1640, when Marcus Banzer wrote his Dissertation on Deafness. Since then there have been many attempts, such as the use of paraffin oil soaked into a cotton ball suggested by Yearsley, wax by Barany, plates by Toynbee, egg skin by Berthold, silver plate by Gompey, cadaver stapes by Politzer and, more recently, corgile (pig's bladder) membrane, collodion and cellophane by Nasiell. The very number of materials indicates the inadequacy of any one material.

The marked improvement in hearing comfort which follows a successfully applied closure of a drum defect is so gratifying and the procedure is so simple for the otologist that any advance in this field is of special importance.

Nasiell in a recently published paper on ear prothesis also emphasizes the importance of a knowledge of ear prothesis for the otologist, and describes at length his technique of applying a cellophane cone to various portions of the middle ear.

During the last three years I have been employing a gold prothesis for the closure of drum defects that has proven of unique value and is possessed of definite advantages over the previously employed methods.

The gold that is used is the cut dental cylinders ordinarily used for small dental fillings, put up in various sizes by several dental supply companies. A cylinder is selected that is slightly larger than the perforation to be closed. Holding the cylinder at the end of a bayonet forcep, it is gently flattened with a second pair of forceps; it is then quickly flamed for sterilization and allowed to cool for a few moments. Through the largest size speculum that fits the auditory canal, the flattened gold cylinder is placed over the drum defect. With a probe, the gold is gently forced into the perforation so that the edges grip the rim of the perforation. It is good to get contact

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Oct. 5, 1939.

GOLD PROTHESIS FOR DRUM DEFECT.

Name	Age	Date	Diagnosis	Results	History
1. F. B.	21	5-12-37	Dry central perforation left eardrum.	Marked improvement in hearing. Perforation closing.	Had otitis, postscarlet, 1921. Hearing impaired left ear.
2. N. B.	43	4-16-38	Perforation left eardrum.	Hearing returned to practically normal.	Perforated left eardrum. Difficulty hearing right ear.
3. R. D.	37	12-12-38	Left perforated drum. Rt. Eustachian tube obstruct.	Fairly good results.	Difficulty in hearing both ears.
4. B. G.	35	5-31-38	Rt. C.S.O.M. Postscarletinal	Improvement.	Difficulty in hearing on rt. ear since childhood.
5. E. I.	43	7-27-38	Perforation left eardrum.	Perforation closed.	Sense of comfort restored.
6. F. J.	10	3-20-37	Perforated left eardrum.	Excellent.	Defective hearing.
7. R. K.	33	6-16-38	Bilateral total drum defect. Rt. chronic suppurative.	Good.	Postmeasle deafness at 3 years.
8. G. L.	39	2-19-38	Left central perforation with thin scar.	Hearing returned to normal.	Noises in left ear.
9. F. L.	22	2-14-38	C.C.O.M.	Hearing returned to normal.	Noises in left ear.
10. M. L.	22	8-30-37	Bilateral chronic catarrhal otitis media. Left eardrum shows thin scar in postsuperior quadrant. Gold cylinder placed to fill scar.	Immediate improvement in hearing.	Defective hearing on both ears.
11. B. N.	53	10-24-34	Perforated left eardrum.	Marked improvement.	Intermittent pain in rt. ear.
12. M. P.	31	2-10-39	Rt. chr. adhesive process. Large central perforation. Left C.C.O.M.	Hearing rt. ear almost normal.	Difficulty in hearing for about 5 years.
13. N. R.	37	5-26-39	Left central perforation.	Good.	Noises have disappeared. Sense of comfort. Burn from acetylene spark.
14. B. S.	34	3-24-37	Bill. chron. adhesive process both ears.	Excellent.	Discharge in right ear. Intermittent since child.
15. R. S.	26	10-20-38	Chronic otitis media suppurative.	Very slight improvement.	Noises at times.
16. B. S.	32	4-13-38	Left C.C.O.M.	Good.	Discharging ear. Right perforation.
17. H. T.	18	8-25-38	Perforated left eardrum.	Hearing returned to normal.	Discharging left ear. Had left mastoid 9 years ago. Had recurrence of discharge 3 weeks ago.
18. S. V.	52	12-28-34	Bilateral perforations.	Results poor.	Defective hearing both sides.

between the gold and the promontory of the labyrinth. Frequently the patient can guide the otologist by stating when the hearing seems best. The change and improvement is usually quite sharp.

The advantages of the spongy gold cylinder are that it is flexible, soft and easily molded, yet is sufficiently light to participate in the drum vibration, a quality not possessed by the silver plates, and it still possesses more permanent form and resiliency than cellophane, cargile membrane or collodion. It is readily sterilizable and nonirritating to the tissues. One of the most satisfying features of its use is the tendency of the gold prosthesis to stimulate epithelialization and closure of large perforations which hitherto seemed improbable, and to lead to rapid closure of small perforations. In cases where hearing was not much impaired, ear noises have disappeared and a sense of comfort in the ear created.

The accompanying series of cases indicates the results obtained with the gold prosthesis. The number improved were as follows: Markedly improved, 56 per cent; moderately improved, 38 per cent; unimproved, 6 per cent. The perforations were closed or closing in two cases.

In some of the cases, where in spite of the presence of relatively good hearing as manifested by the audiogram, even the slight improvement in the audiogram following the prosthesis was followed by an increase in comfort of the patient apparently in excess of the hearing gain.

Prior to application of prosthesis, all suppuration must have ceased.

One can conclude that the use of dental gold cylinders promises a definite advance in ear prosthesis and will contribute much to the practice of the otologist.

32 East 67th Street.

IN MEMORIAM

HENRY LAWRENCE SWAIN, M.D.

1864-1940.

We regret to announce the passing of Dr. H. L. Swain, of New Haven, Conn.

He was the son of George Hussey and Henrietta Swain, both of Nantucket, Mass. His early life was spent in Nantucket until his migration to New Haven in 1873. He was educated in the grammar schools of New Haven; began his training in the Medical College of Yale University in 1881 and received his M.D. degree there in 1884. He spent two years in postgraduate study in otolaryngology in Leipzig, Germany.

For over 30 years Dr. Swain was in active practice in New Haven, and was consultant at the New Haven, Grace, Meridin and Gaylord Farm Sanitarium. For many years he was Professor of Laryngology and Otology at Yale, and in 1935 was named Emeritus in this department.

Dr. Swain was admitted to Fellowship in the American Laryngological Association in 1889, served as Secretary for five years and was President of that organization in 1901.

He was a member of the New Haven City, the County, State, American Medical, American Anti-Tuberculosis and Gaylord Farm Associations. He was an Honorary Fellow of the Laryngological Section of the New York Academy of Medicine.

In 1890, Dr. Swain was married to Miss Etta Winchell.

His gracious personality, his loyalty to his friends, his regular attendance at the meetings of the American Laryngological Association and his co-operation with his laryngological colleagues were outstanding characteristics of the man.

To his widow and his large circle of personal friends we offer our sincere sympathy in his passing.

M. A. G.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLARYNGOLOGY.

Meeting of Oct. 18, 1939.

Surgery of the Auricle. Dr. Gerard H. Cox (by invitation).

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

Oral Plastic Surgery (Abstract). Dr. H. S. Dunning.

(Published in full in this issue of THE LARYNGOSCOPE.)

Some Problems in Nasal Plastic Surgery. Dr. C. R. Straatsma (by invitation).

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

DISCUSSION.

DR. JEROME P. WEBSTER: We have seen some very interesting cases and methods of treatment this evening. Dr. Cox showed some of the difficulties incident to the repair of subtotal and total loss of the auricle. The ear is an intriguing part of the anatomy with which to work, but it takes a great deal of patience and time.

Dr. Cox spoke of using only the skin back of the ear up to the hairline in order to make the anterior portion of the new ear, and of grafting the defect back of that, with a skin graft on the head and one on the flap that has been raised. That is the usual method of building up an ear. It is interesting that Sir Harold Gillies does not limit himself to the use of hairless skin back of the ear, but frequently uses the scalp farther back, and will bring that up on the ear, particularly in subtotal loss of the ear, despite the fact that it bears hair. After he has constructed the ear, he cuts off the hairy skin and Thiersch grafts that area. I haven't used that method myself, but have always used the skin up to the hairline.

Congenital loss of the ear should usually be operated upon very early in infancy. There is very frequently present nothing but a lobe and a small eminence above the lobe. The little nubbin which lies under the skin very frequently contains a considerable amount of cartilage, and if you will operate early on that you may find that there is a very tightly folded bud of cartilage completely wrapped up on itself. You may be able to remove this completely, unfold it, and put it back under the skin that is back of the normal ear. You may later on raise the flap of skin and cartilage and graft on both sides. The advantage of using this cartilage is that it will increase in size with the growth of the patient.

The cartilage of the mother's ear has been used by Gillies, and of course preserved cartilage has been used more recently by Pierce, of San Francisco. I don't think there is much advantage in using the mother's ear over that of using preserved cartilage; however, if you attempt to use the latter in a child, you cannot expect that the cartilage is going to grow with the development of the child; therefore, if you are going to use preserved cartilage it is better to wait until the individual is nearly full grown.

In the outstanding ear, or prominent ear, I would say that it is not always necessary to excise an elliptical portion of the cartilage. There are times when

it is possible to fold the cartilage to form a new antihelix. Most of these prominent ears lack an antihelix, and by thinning out the cartilage along the line of what should be the antihelix and by suturing the cartilage to form a fold, you may be able to simulate the normal antihelix much better than if you merely cut out an elliptical portion of cartilage.

Dr. Straatsma's cases were very interesting and showed some remarkable results. It is very true, as he told you, that one of the difficulties we have in replacing loss of tissue on the nose is to obtain a satisfactory match of the skin. The skin of the cheek and forehead is more like that of the nose than the skin in other parts of the body. It is remarkable, however, that sometimes a Thiersch graft from other parts will assume the natural appearance in color and in texture of the skin of the nose, but the individual characteristics of the patients with whom you are working show great differences. As Dr. Straatsma said, a graft from the inside of the arm matched the surrounding skin very well in a case where such a match was not of great importance, whereas in another case it might fail to match. Individuals may have entirely different types of skin. I recall seeing a case in which Gillies made the vermilion border of the lip in a total reconstruction of the mouth from the very red skin on the neck. It was almost a perfect match for the vermilion border.

Dr. Straatsma wisely advised not to attempt to reconstruct a nose after the removal of a basal-cell epithelioma, or squamous-cell epithelioma for that matter, until a year has elapsed. You would not build a house on top of a fire. To excise an epithelioma and then use your best material in building up a nose, only to have to tear it down again because of the reappearance of the tumor, is a very great tragedy, and you will probably never be able to equal the first result.

Dr. Dunning's paper was extremely interesting. I may say that it brought back days in China when we had untouched cases of diseases causing great deformities. Such diseases as noma and kala-azar were rampant there, with patients often dying of noma. Plastic surgery problems were very great if the patient lived and if the whole half of the face were eaten out. While there I happened to have 13 cases of phosphorus necrosis of the jaws resulting from the use of white phosphorus. It is true that you must be very careful where you have osteomyelitis of the jaw not to remove the jaw unless you have new bone forming. I have taken out the whole mandible from one end to the other in two cases, and the maxillary bones with all the upper teeth. The face did not collapse because the involucrum replaced the dead bone and satisfactory results were obtained.

DR. WILLIAM H. HOLDEN: I very much enjoyed the papers and the discussion which Dr. Webster has just given you. They have covered the field so well that there are only a few points I would like to add.

In the case of Dr. Cox's paper, I think we do have to consider the economic problem of the patient in the reconstruction of some of these losses of the auricle and nose. It is true that in smaller children, babies, and so forth, certain work can be done, such as Dr. Webster suggested to you, in building up these ears. But in cases where older people have lost an ear it is very difficult to get anything that is going to look like an ear. It is going to take a long time to transplant tissue to that part by means of pedicle grafts and in the end have an auricle that looks like anything but an auricle. As an end-result, the patient is usually not very well satisfied. There is a type of prosthesis that has been developed in Germany—a semi-pliable, sponge-like rubber which can be matched very easily with the other ear, and can be applied by means of a special rubber cement. It is almost impossible to tell that the patient has an artificial ear, and they can apply it themselves in just a few moments. It seems to me that rather than subject a patient to the long period of time in the hospital which is necessary to form a new auricle by grafts and transplants, and when the end is not as satisfactory as we would like to have it, it is much better to have a result by prosthesis that looks better. I don't mean that it is not a clever piece of plastic surgery to follow the tech-

nique that we have been shown tonight, but I do think that we must consider the economic as well as the cosmetic problem.

As suggested by Dr. Straatsma's paper, an important factor in nasoplastic cases is the question of obstruction. It is better not to do a submucous resection at the time of the plastic operation, but to attend to this beforehand. Most of these obstructions that are posterior to the anterior third of the nares should be attended to before the plastic procedure. If, however, obstruction involves the columellar cartilage, or if the cartilaginous portion of the septum is twisted to one side, these frequently are better corrected at the time of the plastic procedure.

As mentioned by Dr. Straatsma and demonstrated by his slides, the removal of some of the septum, where it attaches to the columella, will correct the obstruction in cases of dislocation and deformity. But, on the other hand, deformity will often result from the retraction of the columella. I find that better postoperative results are obtained by separating the septum at its dislocated portion from the columella and making a counter-incision back of the deformity through the mucous membrane and cartilage, but not through the mucous membrane of the opposite side. This allows the dislocated portion to fit back into the columella without tension, where it is then sutured. This maintains the support of the nasal tip and columella and at the same time relieves the obstruction.

Dr. Dunning showed us some very amazing results in the field of oral surgery. The only thing I can mention in connection with his work is the combining of nasoplastic work with that of the oral surgeon, because there are so many nasal deformities associated with cleft lips and palates. The correction of these defects, in many instances, must of necessity wait until proper nasal development has taken place before much can be done; therefore, these deformities should be corrected later and not at the time of the oral surgery.

In many of these nasal deformities there is distortion of the nostril caused by the cleft. At the time the lip is repaired it is very difficult not to have some deformity remaining in later years when the nasal structures develop. In these cases, therefore, there is usually marked development of one alar cartilage over that of the other. When the nasoplastic is done these can many times be matched by dissecting out the alar cartilage and sewing them in the midline at the crus. Then one can bring them up in shape so that a fairly good nasal tip results.

Another factor in these cases that is important is the columella. Some of these patients have deformities of the columella that can be remedied by reshaping the nose, such as a case I saw recently for Dr. Dunning, where there was a marked nasal hump. By taking down this excessive development of the nasal bones and reshaping the alar and lateral cartilages, we were able to get a fairly good looking nose. In this particular type, entire reconstruction of the nose was done, including the lateral and alar cartilages. A portion of the nasal septum was resected after it had been separated from its columellar attachment, thus allowing the columella to be raised and assume a normal position.

It is the combination of the nasal plastic work with the oral surgery that is so important, because they are interrelated. Either the oral surgeon does all the work himself, or he works in conjunction with someone doing nasoplastic surgery. I wish to express my appreciation for the papers read tonight. Certainly, they show you the great possibilities of plastic work, and what can be so ably done by these men.

DR. DOUGLAS B. PARKER: It has been very interesting to hear these papers on various plastic procedures. We have, of course, learned a great deal about plastic work since the first World War some 20 years ago, which gave a tremendous impetus to plastic surgery in general.

I am going to limit my discussion to the oral plastic end of it, as brought forward by Dr. Dunning. He showed several cases of cleft palate and cleft lip—and I am glad he used the words “cleft lip” instead of “hare lip.” I hope the time will come when we will all do so. We see a great many of these cases of cleft palate and cleft lip, and it might interest you to know that they occur once in 2,500 births. In a large city like this, we see a great many, not only from our own population but those brought in from outlying parts.

Very few men know when is the best time to operate on these cases. We do the operations in stages. The first stage is closing the lip, and this is done in the first three months. In complete clefts of the palate or lip, premaxillary wiring is frequently done. There are many cases in which premaxillary wiring, while it brings the bones together, has a very damaging effect on the growth of the maxilla, and many of Dr. Brophy's cases that he has demonstrated for so many years had a great deal of malformation due to pressure exerted in the early stages.

I might say that we are doing the soft palate cases much later than we used to. Cases in which there was attempted early closure were almost invariably failures, so we are getting to the point where we believe closure of palates had best be done between ages 2 and 3 years. Of course, if it is done even a little later, we may have still better tissues to work with.

In our first attempt at operation it is so important to see that in each stage we do we get a satisfactory completion of that operation. We so frequently see cases which have been operated on six and eight times by a man unfamiliar with these operations. They continue to break down, and when they come to us they are so full of contracted scar tissue that there is nothing we can do because of inadequate tissue. Every time we lose tissue we lose ground, and the contraction that results is not only bad cosmetically but bad functionally.

Another important point that Dr. Dunning brought out was the curetting of these infected mandibles following cases of osteomyelitis or necrosis of the jaw. I can't emphasize this too much by saying that we find that the jaw is not the same as the long bones of the extremities, such as the femur and tibia and the bones of the arm, in cases of osteomyelitis. In many respects we may find that failure is not due to the actual loss of bone, but to energy of general surgery in prematurely removing bone. Opening up and curetting too frequently has been followed by very bad results. We find that drainage of these osteomyelitis cases—and the term osteomyelitis is used frequently for all sorts of necroses about the jaw—is the essential treatment. By allowing definite new bone to form, it has been attended by very much better results, and we have had many cases where the taking out of an entire jaw was followed by very good regeneration. By taking out the sequestrum as it separated we have had a regeneration of bone, and have achieved a functional, cosmetic looking jaw that otherwise would have been a hideous deformity.

Dr. Dunning also spoke of prognathism, where the lower jaw does not occlude with the upper teeth, due to its prominence. These cases can be corrected by cutting the ramus above the mandibular foramen on a horizontal plane, and sliding the body of the mandible back and stabilizing it with splints or wires on the teeth.

It has been a pleasure to listen to these papers and take part in this discussion.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLARYNGOLOGY.

Meeting of Dec. 20, 1939.

Pharyngomaxillary Fossa Infection. Dr. Raymond J. Gaffney (by invitation).

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

The Use of Colloidal Thorium Dioxide in Roentgenography of Paranasal Sinuses. Dr. Russell C. Grove.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

Meningitis and Temporal Bone Abscess Secondary to Suppurated Petrositis. Clinical Course with Sulfanilamide. Dr. Eugene R. Snyder.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

Cerebrospinal Rhinorrhea. Pathological Findings. Dr. Max L. Som and Dr. Rudolph Kramer.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

Polypoid Tumors of the Esophagus. Dr. John J. Mahoney (by invitation).

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

Two Cases of Esophageal Foreign Body with Complications. Dr. F. Vistreich.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

Osteogenic Fibrosarcoma of the Temporal Bone. Dr. Harold W. Corya.

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

Sphenotemporal Lobe Abscess with an Analysis of Little Known Symptoms. Dr. David L. Poe (by invitation).

(To be published in a subsequent issue of THE LARYNGOSCOPE.)

DISCUSSION.

DR. JOHN M. LORE: I want, first, to congratulate the Chairman on a very well organized program.

About these infections of the neck, I think the thing to bear in mind is to have the courage of one's convictions. When you make a diagnosis of pus, don't be afraid of going after it. As for the question of temporizing, you are taking a chance. I know the feeling is, let it localize and point, but what is going to happen in the interim? You have a closed space with pus in there. Have you any assurance that it is not going to cause thrombosis or spread deeply? The importance of the fascial planes is, of course, sometimes overstressed, giving the feeling that pus must of necessity go up or down, or follow the carotid sheath. Bear this in mind about any suppurative lesion:

Nature throws up a protective barrier. The thing is pretty well walled off, and only when this wall is broken down does the pus spread along the fascial planes.

I want to congratulate Dr. Gaffney for having the courage of his convictions.

DR. E. F. MERRILL: I believe that the use of umbrathor has certain distinctive advantages in studying the nasal accessory sinuses when a contrast medium is necessary. Of course, in many things, like solitary root cysts and retention cysts, we do not need to use an additional contrast medium, but once such a medium is introduced I feel we should not limit ourselves to one or two exposures.

You will notice that Dr. Grove showed films made in several positions, the ordinary posteroanterior, nose-chin, lateral sitting up and lateral films with the patient lying down on both sides, so that we can get the contrast medium as close as possible to the lateral walls. For instance, in the left antrum, with the patient lying on the right side, the contrast medium gives the relief of the nasal wall. Lying on the left side, we get relief of the lateral wall.

Umbrathor is certainly more easily handled than oil, although we now have iodized oil put out in two degrees of viscosity. The first, lipiodol, was quite unhandy to use in a small syringe. We now have a lighter oil, but it is not as handy to use as a liquid medium. We find that the contrast medium, umbrathor, stays in the spaces quite long enough. Some apprehension might be felt about that but we find it does stay as long as necessary. In fact, if introduced into soft tissues it is not absorbed for several weeks, or even longer. The relief qualities offer certain advantages because a thin layer of umbrathor is not as radiopaque as a correspondingly thin layer of oil.

(To be continued in a succeeding issue.)

BOOK REVIEWS.

Curietherapie des Tumeurs Malignes des Voies Aeriennes et Digestive Superieures. By Prof. C. Torrigiana, Directeur des Services O.R.L., des Hopitaux reunis de Florence; and Prof. V. Palumbo, Directeur de l'Institute radiotherapique Italien de Florence. With 175 pages and 68 illustrations. Bordeaux: Editions Delmas, 6 Place Saint-Christoly. Paper. 1939.

The authors relate their personal observations during 25 years of collaboration. According to their present knowledge, the keystone in the radiotherapeutic system is dosing. The authors describe the physical and pathological anatomy. They emphasize the need of collaboration between the clinicians of the various branches and the radiotherapists. This collaboration should continue throughout the entire treatment. The authors feel that "we must employ larger surgical proceedings and radiotherapy of greater intensity than the clinical data would lead us to consider sufficient."

In detail the fundamental problems of surgery and radiotherapy are taken up; namely, tumors, lymph nodes and recurrences. The treatment of tumors in various localities in the field of otolaryngology is described, both surgery and irradiation. The monograph has 175 pages, with 68 illustrations.

A well composed work and should be in the library of every otolaryngologist interested in cancer.

H. B. O.

Cancer of the Larynx. By Chevalier Jackson, M.D., Sc.D., LL.D., F.A.C.S., Honorary Professor of Broncho-Esophagology and Consultant in Broncho-Esophagologic Research, Temple University Medical School, Philadelphia; and Chevalier L. Jackson, A.B., M.D., M.Sc. (Med.), F.A.C.S., Professor of Broncho-Esophagology, Temple University Medical School, Philadelphia. Three hundred nine pages with Index. With 189 illustrations on 116 figures and five plates in color, containing 50 illustrations. Philadelphia: W. B. Saunders Co. 1939.

An entirely new book dealing with the operative and nonoperative treatment of cancer of the larynx. The authors have arranged the book in three parts. Part One takes up early diagnosis, and each diagnostic procedure is discussed in detail. The various operative procedures—laryngofissure, laryngectomy and irradiation—are evaluated. Postoperative complications and how to avoid them is given adequate discussion.

Part Two concerns the general consideration of laryngeal cancer, as to etiology, pathology and prognosis, with many tables showing the results of treatment by laryngofissure, laryngectomy and irradiation.

Part Three deals with the history of laryngofissure, laryngectomy and irradiation in the treatment of laryngeal cancer. The monograph is well illustrated with many illustrations in color by Dr. Chevalier Jackson. It also contains a large bibliography.

H. B. O.

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